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PELLAGRA IN THE UPPER INDIA PROVINCES

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Pellagra exists and prepellagrous state is common in the Upper India provinces. Cases have been reported from Bengal, Bihar, the U.P. and the Punjab but none from Baluchistan, North West Frontier Provinces, Sind, Rajputana, the Central India and the Central Provinces. We often see the cases in our every day practice but their true nature is very seldom detected. A recapitulation, therefore, of the facts regarding pellagra with a view to facilitate the diagnosis and treatment of it in general practice is, to my mind, very desirable and necessary.

ÆTIOLOGY

Ætiology of pellagra, in spite of the great researches and advancements of knowledge, is not yet clear nor definite. Many attractive theories have been put forward at various times and one could examine them only to find where they err. The peculiarities that I have noted and the conclusions that I have drawn relating to the ætiological factors concerning pellagra patients in India are briefly set down.

1. Pellagra occurs in epidemic and endemic forms in maize-eaters in Rumania, Italy, Spain and in certain other countries of Europe and in those in America who use alcohol prepared from stale and rotten maize. Thus pellagra is invariably associated with maize in those countries. In India, Batra (1942) has also reported pellagra in Bilaspur State and in Simla Hills where maize is the staple food. It has also been noted that the pellagrous state is often associated with certain chronic exhausting ailments in sufferers who are not maize-eaters. Pellagra is thus described as:—(*a*) Endemic pellagra, (*b*) alcoholic pellagra, and (*c*) pellagra secondary to organic diseases.

A careful study of the available literature in India and of the cases that I have had the opportunity to see and study in the U. P. and Bihar affords a clear indication that pellagra in epidemic and endemic forms occurs only in maize-eaters and in those who use alcohol fermented from pellagra-producing maize and that sporadic cases which we get in the U.P. and Bihar and for that matter anywhere in the plains of India are mostly secondary to some other organic diseases and are independent of maize-eating.

Is the modern maize-toxin theory applicable to cases in India? The most modern accepted theory as to the probable causation of pellagra is that a special toxin in the presence of nicotinic acid deficiency causes the damage. This theory assumes that this special toxin is a maize-toxin (Chick, 1933) but as we now definitely know that certain chronic exhausting diseases and malnutritions could also produce pellagra we have to assume that they are also capable of producing in the body a particular type of maize-toxin-like substance which is capable of causing secondary pellagra. This certainly is not a very feasible assumption. The maize-toxin theory is, any how, not true for most of the Indian patients as maize is not their staple diet. How-

ever, as the nature of this pellagra producing toxin, if it exists at all, is not yet known to us we leave the discussion of it at this stage.

What cases should be called as those of pellagra? In a case of rickets which is a deficiency disease we call it rickets whether all or some particular signs and symptoms of it get manifested or not. We now know for certain that nicotinic acid deficiency in the body is essential for the causation of pellagra but this deficiency does not always produce the characteristic rough and pigmented skin as an initial manifestation of its existence but may give rise to only glossitis, general inflammation of the mucous membranes or to definite gastrointestinal, circulatory or mental disturbances. The question, therefore, that could pertinently be asked is:—Are we justified to call a patient having any portion of the above described symptom-complex amenable to nicotinic acid as a case of pellagra or should we label it as such only when skin manifestations characteristic of pellagra are present? However, as the question is still unsettled I call a case as that of pellagra when characteristic skin lesions are present and label the whole symptom-complex minus the skin changes as prepellagrous state.

Extent of the prevalence of prepellagrous state in India—Joseph (1941) working in Bombay Presidency found that, at one time, out of all the 82 in-patients in the medical department of the Miraj Medical Centre, 50 had porphyrinuria and 34 of the 50 were found to have some signs of prepellagrous state and a careful clinical examination, keeping pellagra in mind, revealed that 41 per cent of the total medical indoor patients had one or more symptoms of prepellagrous state and that 47 per cent were found positive to pellagrous state by therapeutic test with nicotinic acid and yeast. This interesting clinical experiment confirms my belief and shows in a clear manner how very prevalent nicotinic acid deficiency causing pellagrous and prepellagrous state is in Indian patients.

Clinical Course

Four systems of the body, cutaneous, gastro-intestinal, circulatory and nervous, are generally the seat of the manifestations of pellagrous lesions. They may be involved singly or severally. The onset may be febrile or afebrile and very insidious when only the cutaneous system is involved but grave constitutional disturbance may set in when the gastrointestinal and circulatory systems are attacked from the very start. The mental state from the beginning or at any stage of the disease may become actually alarming. Nervous lesions when present are generally due to associated B_1 deficiency. Three illustrative cases accompanied by photographs are cited with a view to present a clear picture of the clinical course of the disease.

Case 1—Male, 45 years, on duty at Mussoorie, got diarrhœa and vomiting, diagnosed as cholera. Vomiting stopped but six to eight motions a day, resisted out-door and in-door treatment at various places; lost 20 lbs. in weight. Skin lesions appeared, were diagnosed as eczema and treated without relief. Tried Ayurvedic cure for sprue for 1½ months; lost 72 lbs. and then treated for colitis for 3 months with torantil, abidol, campolan *etc.;* improved and resumed work at Hardwar after one year in July 1937. Had a relapse after two years in March 1939; tried old remedies but to no avail. Became positively mental; refused all treatment; wanted to resign service. Admitted to Railway Hospital, Moradabad, January, 1940, was treated with campolan, betaxin and various other drugs with no response. Diarrhœa with at times 30 to 40 motions a day resisted all remedies. I was shown the case in February, 1940.

Condition on examination—He was in the terminal stages of the disease. Had dyspnœa, rapid pulse, dilated heart with bruit; swelling of the whole body, symmetrical scaly pigmented skin of legs. B.P. 92/50. Total W.B.C. 5312, R.B.C. 2,923,500; Hg. 90%; colour index 1·5 and a blood picture of megalocytic hyperchromic anæmia. He was thin, weak and emaciated with rough and mottled skin of the abdomen and chest (Fig. 1). I diagnosed him as an advanced case of pellagra.

Treatment and result—He was put on nicotinic acid and gradually increased high protein diet. I left Moradabad and my colleague later on informed me that his recovery was dramatic. He began to digest his food in a few days. Diarrhœa was controlled. B.P. rose to 110/70. Oedema disappeared, heart became normal, mental state changed, W.B.C. increased to 13,125, R.B.C. 3,157,000 and all that in 20 days. He regained 42 lbs. weight in two months and joined his duties on 11-4-40. I saw him on 14-5-40 and he complained of 2 to 3 motions a day which resisted ordinary treatment. 'Ispaghoul' seeds with curd (*dahi*) advised, which controlled the motions in 2 days. I saw him again at Hardwar on 18-7-40 and found him in sound health (Fig. 2). Slight dermatitis at the region of the chest persisted for which I advised sodium thiosulphate. His usual diet was wheat-phulkas, dal, rice, vegetables and tea but never maize and I added eggs, liver and a pint of raw or lightly boiled milk to it.

Case 2—On 7-6-40 a man saw me with a view to find out if I could help his brother who had peculiar ailments for last one year which resisted all treatments and who was almost getting maniacal and often talked of ending his wretched life.

Previous History—A railway fireman of Moradabad, unmarried, aged 23 years, had painful micturition. Noted dark patches on his face a year ago and then began to feel tired after work; lost appetite, had fleeting aches in the body and troublesome pains in elbow, knee joints and severe cramps in calf muscles. Began to worry over simple things, got "puzzled

FIG. 1—Showing general condition before nicotinic acid therapy. (Case 1).

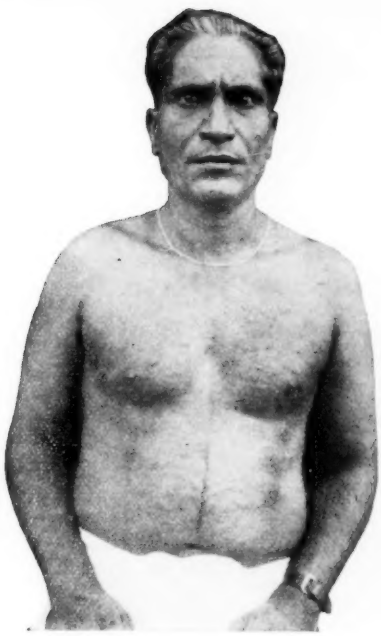

FIG. 2—Same case as in Fig. 1 after nicotinic acid therapy.

FIG. 3—Showing 'butterfly patch' on face and rough pigmented skin of forehead, temples, lips and chin. (Case 2).

FIG. 4—Same case as in Fig. 3 after nicotinic acid therapy.

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FIG. 5—Same case as in Fig. 3 showing symmetrical pigmented lines on toes due to friction and pigmented and rough skin of hands.

FIG. 6—Same case as in Fig. 3 after nicotinic acid therapy.

FIG. 7—Showing 'collar patch' on neck and erythematous patches on fingers and scaly patches on nose, chin and temples. (Case 3).

FIG. 8—Same case as in Fig. 7 after liver and nicotinic acid therapy.

headed", lost all interest in life, was morose and taciturn. Indigestion, flatulence, pain in abdomen and constipation were troublesome but there was no diarrhœa. A Hakim restricted his diet to bread and 'Dal Mung' for three months which increased his troubles and made him a definite *mental* case with suicidal tendencies. He was diagnosed and treated during the year for gonorrhœa, chronic malaria, syphilis, dyspepsia, gastric ulcer, deranged liver and 'heated blood' and was also taken for a malingerer and later on the doctors took no notice of his troubles and declared him a neurasthenic and melancholic patient.

Condition on Examination—There was pellagra written on his face. There was a rough pigmented 'butter fly' patch on his face and rough pigmented scaly skin of forehead, lower lip and chin and 1½"×1½" hard and almost black patches on his temples (Fig. 3). There were dark, small, circular patches of the extensor surface of his forearms and hands and thick black patches with a scaly core in the centre at the roots of the index and middle fingers on the left and middle and ring fingers on the right. The dorsum of all the toes of his feet had symmetrical lead-colour pigmented lines (Fig. 5). There was also slight mottling of his back.

Treatment and result—I prescribed nicotinic acid with the addition of meat and liver soup, marmite and milk to his daily diet and left the place. The response to the treatment as described by the doctors and the patient himself was nothing short of marvellous. The skin condition on the hands, forearms and back altogether disappeared and the patches on the face and toes became smoother and lighter in colour after the first injection. The pain in joints and muscles disappeared and the patches on the face and toes became smoother and lighter in colour after the first injection. The pain in joints and muscles disappeared and the man began to feel hungry after the fourth injection. He became mentally normal on the 5th day but as soon as the nicotinic acid was stopped the pigmentation and roughness reappeared in the patches on his face. I examined him after 40 days and found him greatly improved (Figs. 4 & 6). I again saw him after 15 months and found him in perfect health. Skin lesions were gone except the dark and rough patches on the temples. He still takes liver soup.

Case 3—A fair skinned girl aged 5 years showed aversion for food, was easily excitable, had disturbed sleep and was considered to be a backward child; had pain in the abdomen and head during last winter and bouts of diarrhœa and constipation all along till one morning in April, 1940, her mother noticed small circular red patches situated in a continuous line on the side of her neck just above the collar of her frock. Her lips became blue, dry and cracked. The child was taken to Mussoorie. Two days after arriving at the hills on 8-6-40, red, angry looking, erythematous patches appeared in between the fingers of her hands. There were scaly patches on the tip of her nose, temples and chin (Fig. 7). She also had an attack of diarrhœa. I saw her and first thought of solar dermatitis and later on, of pellagra. The 'collar patch' on the neck became completely black. I put the child on calf-liver soup, marmite, virol and two half boiled eggs a day, and gave nicotinic acid from the 12th day. Her skin condition began to clear from the 3rd day. The fingers were alright in a week and the black collar patch disappeared in a fortnight (Fig. 8). Her general and mental condition also began to improve and she behaved as a normal child by the 25th day.

Relapses—I followed the case all along. 2 years after the 1st attack, in April, 1942, she had a recurrence at Cawnpore. She lost her appetite and again began to show aversion for food. Then the "butterfly" patch on the face and skin lesions on fingers and shins appeared. Nicotinic acid cleared the whole thing in a week. Two months later another relapse with severe dermatitis on dorsum of her feet occurred. Nicotinic acid cleared it and a maintenance dose is now continued.

Comment

1. Maize is not the staple diet of any of my cases showing that maize-eating is not an essential factor in India.

2. The cause of pigmentation and roughness of skin in the 1st case was ordinary sun-exposure, in the 2nd case infra-red rays from the "fire-box" off the engine falling on his temples and face and the friction of rough boots on his toes and in the third case actinic trauma due to intense ultra-violet rays of the sun acting in a dry and cool air of Mussoorie, showing that any severe and continuous trauma or friction may cause skin lesions in a patient having nicotinic acid deficiency and that sun-rays are not the sole cause.

3. Severe gastrointestinal and other constitutional disturbances were present in the first case, marked mental symptoms in the second and prominent skin lesions in the third, showing the great variety of symptoms that pallegra cases may present.

4. The cases, once correctly diagnosed, respond to nicotinic acid therapy marvellously but there is danger of relapses if the patient is allowed to go back to his previous way of living and maintenance doses of yeast, liver or nicotinic acid are not continued for years.

Diagnosis

A careful study of some of my cases has proved to me that it often becomes very very difficult specially in cases exhibiting grave constitutional disturbances to say whether a case is primarily of pellagra and need no more treatment for any other condition or the pellagrous symptom-complex is simply an associated condition developed on a case of some other disease which should be treated in spite of the dramatic improvement produced by the administration of nicotinic acid. For the purpose of diagnosis, therefore, the cases met with in India may be grouped into the three following heads:

1. Typical cases—very easily diagnosed if pellagra is kept in mind.

2. Doubtful cases—not easy to diagnose unless special attention is paid to the condition and a painstaking previous history taken and a very careful examination made by the attending physician.

3. Difficult cases—when skin lesions are not present in the beginning only nicotinic acid test may give the clue though an experienced clinician can get much out of the history, clinical course and the degree or response to the treatment of the apparent malady and by testing urine for porphyrinuria.

Typical cases—They invariably show characteristic pigmentation of the skin, some gastrointestinal disturbance, low blood pressure and some form of anæmia. Glossitis and diarrhœa may or may not be present. Diminution or absence of free gastric HCl is the rule but excess of it was also noted in one of the recorded cases. The anæmia may be micro- or macrocytic and colour-index high or low. Mental syndrome when present is definite, well-marked and oppressing. It, therefore, comes to this that in order to avoid missing a typical case one should keep pellagra in mind and make it a rule that whenever one meets an obstinate case of gastrointestinal trouble or an intractable one of anæmia or a case showing definite mental disturbance without any apparent cause one should invariably examine him in a good light to see if he shows some skin lesions and take his blood pressure and, if possible, examine his urine for porphyrin.

Doubtful and difficult cases—In order to diagnose such cases the following procedures may be adopted: 1. A history of previous and present illness is taken eliciting specially the modes of onset and any likely periodicity of the attacks, any symptoms of indigestion, flatulence, chronic diarrhœa and loss of appetite, vague abdominal pains, cramps and twitchings in the legs and other muscles and the mental attitude of the patient towards his illness and environments. 2. A very careful examination of the skin and buccal mucosa is made in a very good light specially noting the texture and colour of the skin and any dry, scaly, pigmented patches and signs of dermatitis on it or glossitis, angular stomatitis and pigmented mucosa of the inner sides of cheeks, sides of tongue and inner parts of lips and a rough and scaly pigmented "butterfly patch" on the nose and cheeks which may have formerly been taken to be due to bad liver. 3. Three special examinations are then made: (*a*) Blood pressure readings taken at morning, noon and evening. (*b*) Urine tested for porphyrinuria. (*c*) Blood tested for anæmia and its variety. In connection with the variety of anæmia and the size of red blood cells I have to sound a note of warning against Eve's halometer which often caused great upsets in my otherwise careful observations till Chuttani (1942) conclusively proved it to be a thoroughly useless and unreliable instrument.

The interpretation of the investigation may be on the following lines: (1) Porphyrinuria and anæmia present, blood pressure low (systolic below 100 mm. Hg.) and either history is suspicious or even a single symptom of prepellagrous state is present, nicotinic acid deficiency is a certainty. (2) No arrangement for testing porphyrinuria but anæmia is present, blood-pressure is very low and either history is suspicious or one or more of the prepellagrous signs are present, nicotinic acid deficiency is a probabiity. (3) No arrangements for testing porphyrin in urine and blood for anæmia but blood pressure is very low, patient an ever-ailing or chronic sufferer from gastrointestinal troubles, anæmia and weakness of unknown origin, or has vague mental and nervous conditions or insignificant-looking, dry, scaly, pigmented skin lesions or is having some such other seemingly ordinary disease which is proving intractable to routine treatment, nicotinic acid deficiency is suspected and therapeutic test with yeast and nicotinic acid is justified and should be employed. If some such method of investigation be followed one will be astonished at the number of cases classed as ever ailing chronic sufferers or never-to-do-well under treatment clearly revealing their true nature and amazingly yielding to the correct additional therapy.

Value of porphyrinuria in the diagnosis of pellagra—It has been asserted that porphyrinuria is an integral part of pellagra. Joseph (1941) recently made an enquiry into the question. He found that 61 per cent of the 82 in-patients of the Miraj Hospital observed had porphyrinuria and 87 per cent had some other diseases causing the condition. Caproporphyrin in traces is a normal constituent of urine but it is excreted in larger amounts in diseases affecting liver and the gastrointestinal tract, phthisis and other cachectic conditions. Then, there is uroporphyrin giving port-wine colour urine which is toxic in origin and is due to prolonged use of sulphonal group of drugs. A familiar type is also known which produces idiosyncrasies to ultra-violet rays and

is a dread of ray therapeutists. It, therefore, appears that the presence of more than traces of porphyrin pigments in urine could generally be found in those cases of pellagra who suffer from more or less severe constitutional disturbances but it cannot be said with certainty whether the urine of those pellagra patients who have only skin lesions and no constitutional disturbances would invariably contain porphyrin or not. The value of the test is thus limited. We may, therefore, conclude that porphyrin test when positive in a suspected case of pellagra having constitutional disturbances may help to clinch the diagnosis of pellagra, that porphyrin test when negative in a suspected case of pellagra having constitutional disturbances definitely excludes pellagra and that porphyrin test when negative in a suspected case of pellagra having only skin lesions and no other trouble has no value as it is not known whether pellagra cases having only skin lesions have excess porphyrin in their urine or not.

Treatment

It is dramatic with nicotinic acid and is also successful without it if yeast and protein diet of high caloric value could be given. The treatment of the primary condition—a chronic malady or a maltnutrition—should not be neglected. I give 50 to 100 mgm. of nicotinic acid in 20 c.c. of 15 per cent glucose intravenously daily, on alternate days or twice a week according to the gravity of a case for 6 to 12 injections or till improvement in grave symptoms is definite and clear. I, then, give four to six tablets orally, *i.e.*, 200 to 300 mgm. per day till the patient is cured and then give maintenance dose of 100 mgm. daily for a week, then on alternate day in the 2nd week and then twice a week and later on give only liver soup once a day for months and years as an extra to the normal balanced diet. Iron for micro- and liver for macrocytic anæmia and vitamin B_1 for nervous lesions like paresis and neuritis and symptomatic treatment for other symptoms all become essential. When nicotinic acid and expensive vitamin B_1 are not available, cases could be treated very successfully with yeast and sprouted grams or wheat germs and a balanced diet. I give medicinal yeast in 60 grains and wheat germ in 5-ounce doses a day. I also add a cup of liver juice prepared in a steam-cooker to two principal meals and dilute HCl 1 dram before and iron and arsenic tonic after meals. Skin condition also improves with this treatment but

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PROTHROMBIN TIME IN PULMONARY TUBERCULOSIS

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During the last four or five years, with the discovery of vitamin K by Dam and his co-workers and the subsequent animal experiments and clinical observations leading to a better understanding of the coagulation anomalies and hæmorrhagic conditions, many new studies have been undertaken to estimate the prothrombin content of human blood in health and disease by various techniques. One of the earliest and also widely used method is that of Quick (1938) familiar now to all laboratory workers and to many clinicians, both through the papers of Quick himself as well as through many others who effected some slight modifications in the test. While most of the papers have already established a definite relation between prothrombin deficiency, liver injury, liver disease, and biliary tract disease, a few workers found some variations from normal in different nutritional conditions in toxæmias and infections. The authors of this paper, while studying the prothrombin time in normal adults and in different clinical conditions, were naturally prompted to investigate whether there was in addition to all the other explanations already given, any coagulation defect in cases of pulmonary tuberculosis where hæmoptysis was a common manifestation.

We decided to utilise the clinical material available at Vizagapatam to examine a few cases of tuberculosis for prothrombin deficiency, according to Quick's method (Quick *et al*, 1935). In all, we examined 31 indoor and 5 outdoor patients. The results are presented in a tabular form at the end of this paper.

On examining the data collected, the first impression was that prothrombin time is not markedly prolonged in the entire series. A detailed analysis was then attempted regrouping the cases into the following categories:

I. *Cases with hæmoptysis after treatment*—There were 8 cases which had hæmoptysis on admission to the Hospital (No. 1-5 and No. 11-13). In cases No. 1-5,

the prothrombin time was less than in control blood. These cases, received just a few days before the test, hæmostatic serum or Congo red or both and were on good diet. Cases No. 11 to 13 showed prothrombin times slightly longer than that of the control blood. One out-door case which gave history of hæmoptysis sometime previously and put only on calcium lactus, gave a prothrombin time of 24 seconds, while the control gave 20 seconds.

II. *Cases with hæmoptysis and without A. P. treatment*—There were 5 such cases among the in-patients (No. 14, 17, 22, 25, 26, 30 and 31) and 3 among out-door patients. (O. P. No. 2, 3 and 4). Cases No. 17, 22, 25 and 31 showed prothrombin times shorter than the control blood. Case No. 14 had a prothrombin time of 23. No O. P. case gave a prothrombin time shorter than the control, while the other two gave irregular time than the control. Apart from the differences in the clinical condition, the only common feature of all the in-patient cases was that they were on good diet whereas the diet of the out-door cases is generally below the standard requirements. Even with the ordinary diet of lower classes the patients did not show any marked prolongation of prothrombin time. The number of observations, however, is too small to admit a generalisation.

III. *Cases without hæmoptysis but after A. P. treatment*—There were 12 such cases (No. 6, 8, 9, 15, 18-21, 26-28 and 30). Of these cases, No. 6, 15, 20, 21, 26 and 30 gave prothrombin times shorter than the control. No. 8 and 9 gave prothrombin times equal to that of the control. No. 19 gave a prothrombin time slightly longer than that of the control. In view of the recent experimental work suggesting that the lungs are the seat of destruction of prothrombin the shorter prothrombin times in cases with A. P. treatment seemed to require further study.

IV. *Cases with a history of hæmoptysis*—Of these, case No. 24 with a history of spitting of blood showed a prothrombin time of 19 seconds and among the out-patients No. 1 gave a prothrombin time of 24 seconds (control 20 seconds), while No. 5 gave 23 seconds (control 24 seconds).

V. *Last group includes a variety of conditions*—Case No. 19 was one of secondary deposits in the lung and with a suspected cavity in the left side. This gave a prothrombin time slightly longer than the control. No. 23 was a case with a cavity in right lung on which thoracoplasty has been done and the prothrombin time in this case was shorter than that of control. Case No. 24 with a history of spitting of blood developed spontaneous pneumothorax of the left side and had infiltration on the right side. It was put on simple mixture, but was on good diet. The prothrombin time was shorter than the control. Case No. 29, an old case of thoracoplasty, gave a slightly longer time than the control.

Only one case in the whole series (No. 11) was put on vitamin K, given intramuscularly once only. The medical officer in charge reported that no perceptible improvement was noted even after the injection, as the patient continued to have hæmoptysis. One hesitates to draw any conclusions from a stray and single observation on a single case.

(*Continued from page* 5)

does not totally clear up when in a chronic state. It generally takes years to get rid of the long standing rough and pigmented patches of the skin whether sodium thiosulphate is given to clear them up or not.

Prognosis

An undiagnosed case when once the constitutional disturbances start often ends fatally and even a mild mental case may commit suicide at any time; but if diagnosis could be made even in the very late stages of the disease and nicotinic acid is available and could be given intravenously, the case invariably gets over the trouble and could actually be snatched from the claws of impending death. With right diagnosis and yeast and nicotinic acid therapy cent per cent cures could be guaranteed but recurrences would occur if patients are allowed to revert to their old conditions.

Acknowledgment

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TABLE SHOWING THE CLINICAL CONDITION AND PROTHROMBIN TIMES IN TUBERCULOSIS PATIENTS

Serial No.	History	Clinical condition	Treatment	Diet	Prothrombin time in seconds	Remarks
	In-patients:					
1.	Cough, loss of weight, loss of appetite—10 months' duration.	Infiltration right lung, (cavity?), hæmoptysis on 3 occasions in 8 days, 3 ozs. each time.	A.P. (right), Calcium gluconate, Congo red, hæmostatic serum.	Convalescent milk and bread diet, batavias.	18	Thromboplastin 10 days old. P.T. of control blood—21".
2.†	Cough, blood in sputum, fever—15 days' duration.	Abscess lung (?) hæmoptysis on 2 occasions, 2 ozs. each time.	Calcium gluconate, Congo red, hæmostatic serum.	Convalescent milk and bread diet, eggs, batavias.	16	Ditto.
3.	Cough, blood in sputum —duration 3 months.	Bilateral pulmonary tuberculosis, cavity left lung, slight hæmoptysis.	A.P. (left), Calcium gluconate.	Convalescent milk and bread diet, eggs, sweet limes.	18	Ditto.
4.	Fever, cough, blood in sputum.	Bilateral pulmonary tuberculosis, right lung more involved, had hæmoptysis once, 2-3 ozs. blood.	A.P. (right), Calcium gluconate, Congo red.	Do.	21	Ditto.
5.	Cough, fever, blood in sputum.	Infiltration left lung, hæmoptysis twice—once at home, 2 ozs. each time.	A.P. (left), Congo red.	Rice with mutton, vegetables, fruit, milk, bread.	20	Thromboplastin 1 day old. P.T. of control blood—21".
6.	Cough, pain in left chest, loss of weight—duration 10 months.	Bilateral pulmonary tuberculosis.	A.P. (right) partial, followed by A.P. (left) partial.	Convalescent milk and bread diet, sweet limes, eggs.	18	Ditto.
7.	Fever, cough, expectoration, blood in sputum, pain in chest—duration 15 days.	Bilateral pulmonary tuberculosis blood streaks in sputum.	Calcium gluconate.	Rice, mutton, vegetables, eggs, batavias, milk.	18	Ditto.
8.	Fever and cough—6 months' duration.	Tuberculosis right lung.	A.P. (right).	Convalescent milk and bread diet, eggs, sweet limes.	20·5	Ditto.
9.	....	Infiltration and cavitation right lung.	A.P. (right), Calcium gluconate.	Convalescent milk and bread diet, sweet limes.	21	Ditto.
10.	Cough, expectoration—5 months' duration.	Cavity right lung.	Calcium gluconate.	Rice, milk, bread, sweet limes.	23*	Thromboplastin 2 days old. P.T. of control blood—21·5".
11.	Fever, and cough—1½ months' duration.	Cavity right lung, hæmoptysis twice on successive days.	A.P. (right), Calcium gluconate, Congo red, hæmostatic serum, vitamin K.	Convalescent milk and bread diet, eggs, batavias.	22	Ditto.
12.	Fever 4 months' duration, blood in sputum.	Cavity right lung, hæmoptysis once.	A.P. (right), Calcium gluconate, Hemoplastin.	Convalescent milk and bread diet, batavias.	24*	Ditto.
13.	Cough, fever, wasting, loss of appetite—duration 1 year.	Bilateral pulmonary tuberculosis, mild hæmoptysis once.	A.P. (right), Calcium gluconate.	Convalescent milk and bread diet, eggs, sweet limes.	24*	Ditto.
14.	Cough and pain in chest, 5 months' duration.	Bilateral pulmonary tuberculosis with cavitation.	Calcium gluconate.	Rice, coffee, bread.	23*	Ditto.
15.	Cough, loss of appetite, sleeplessness, 6 months' duration.	Tuberculosis, right lung.	A.P. (right).	Convalescent milk and bread diet, sweet limes.	19·5	Ditto.
16.†	....	Pleurisy right, pneumonia?	Calcium gluconate.	Convalescent milk and bread diet, sweet limes, eggs.	23*	Ditto.
17.	Fever and cough, 4 months' duration.	Bilateral pulmonary tuberculosis, cavitation left.	Calcium lactate.	Rice, bread, milk, eggs, sweet limes.	19	Ditto.

* Prolonged prothrombin time due to moisture in tube.
† Cases of non-tuberculous affections of the lungs, admitted into Tuberculosis Wards.

Table showing the clinical condition and prothrombin times in tuberculous patients—Contd.

Serial No.	*History*	*Clinical condition*	*Treatment*	*Diet*	*Pro-thrombin time in seconds*	*Remarks*
18.	Fever and cough, 2 years' duration.	Bilateral pulmonary tuberculosis.	A.P. (left), Calcium gluconate.	Convalescent milk and bread diet, eggs, batavias.	19	Ditto.
19.†	Cough and fever, 3 months; blood in sputum; radium treatment for cancer cervix 1 year back.	Secondary deposits left lung?, (cavity left?), hæmoptysis in small quantities on two occasions.	Calcium gluconate.	Convalescent milk and bread diet.	23*	Thromboplastin 2 days old. P.T. of control blood—21·5″.
20.	Fever and cough, 2 years' duration.	Bilateral pulmonary tuberculosis.	A.P. (left), Calcium gluconate.	Rice, milk, bread, sweet limes.	19	Ditto.
21.	Cough, pain in chest, 1 year.	Tuberculosis left lung.	A.P. (left), Calcium gluconate.	Rice, vegetables, bread, butter, batavias.	17	Thromboplastin 5 days old. P.T. of control blood—20″.
22.	Cough and evening rise of temperature, 2 months' duration.	Tuberculosis right lung.	Calcium gluconate.	Convalescent milk and bread diet, rice, vegetables.	18	Ditto.
23.	Cough, fever and expectoration, 1 year duration.	Cavity right lung.	Thoracoplasty.	Rice, bread, milk, batavias.	18	Ditto.
24.	Cough, breathlessness, spitting of blood, 6 months' duration.	Spontaneous pneumothorax, left. Infiltration right lung.	Mist. T.P.S.	Convalescent milk and bread diet, eggs and batavias.	19	Ditto.
25.	Fever and cough, 2 months' duration.	Tuberculosis right lung.	Mist. T.P.S.	Rice, coffee and bread.	16	Ditto.
26.	Fever and emaciation, 2 months' duration.	Bilateral pulmonary tuberculosis.	A.P. (left).	Convalescent milk and bread diet, batavias.	19	Ditto.
27.	Fever and cough, 1 month duration.	Infiltration right lung and cavity formation.	A.P. (right).	Convalescent milk and bread diet, eggs and batavias.	22	Thromboplastin 9 days old. P.T. of control blood—20″.
28.	Fever and cough 4 months' duration.	Tuberculosis left lung, commencing infiltration right lung.	A.P. (left).	Convalescent milk and bread diet, rice and batavias.	20	Ditto.
29.	Old case of thoracoplasty right side.	Infiltration right lung base.	Calcium gluconate.	Rice, milk and bread, eggs and batavias.	22	Ditto.
30.	Cough and fever, 15 days' duration.	Tuberculosis left lung.	A.P. (left), Mist. T.P.S.	Rice, sago, kanji, milk and fruit.	19	Ditto.
31.	Cough and fever, 6 months' duration.	Tuberculosis bilateral.	Mist. T.P.S.	Convalescent milk and bread diet, eggs, sweet limes.	19	Ditto.
	Out-patients:					
1.	Cough, expectoration, hæmoptysis, and fever 1 month.	Bilateral pulmonary tuberculosis with cavitation.	Calcium lactas. Mist Sed. Expec.	——	24	Thromboplastin nine days old. P.T. of control blood—20″.
2.	Cough, expectoration and wasting, 6 months' duration.	Bilateral pulmonary tuberculosis with cavitation.	Ditto.	——	25	Ditto.
3.	Cough, expectoration and fever, 2 months' duration.	Bilateral pulmonary tuberculosis.	Ditto.	——	25	Thromboplastin one day old. P.T. of control blood—20″.
4.	Cough, 1 month.	Incipient bilateral pulmonary tuberculosis.	Ditto.	——	20	Ditto.
5.	Cough, weakness, fever, blood in sputum, 4 months' duration.	Bilateral pulmonary tuberculosis.	Ditto.	——	23	Ditto.

* Prolonged prothrombin time due to moisture in tube.
† Cases of non-tuberculous affections of the lungs, admitted into Tuberculosis Wards.

Results and Conclusions

Eleven out of thirty-three tuberculosis patients showed some prolongation of prothrombin time. Nine showed only a slight prolongation, which according to our previous data, corresponds to 65 to 80 per cent of prothrombin in the blood. This degree of lowering is not sufficient to give rise to bleeding or to be taken as a warning of coming danger. It is generally agreed that normally the prothrombin in the blood is in excess, and only when it falls to as low a level as about 30 per cent does tendency to bleed actually occur. A few patients who had hæmoptysis a day or two prior to the tests, showed normal prothrombin time. Hæmoptysis in these cases may not have been due to fall of prothrombin or even if it were the case, the subsequent rest and treatment and bodily reaction might have combined to restore the normal prothrombin level. Only two patients showed a prolongation of prothrombin time, corresponding to 50 per cent prothrombin in blood. These being out-patients and belonging to low class families, it is difficult to judge whether the prothrombin deficiency is entirely due to nutritional defects or toxicity of the disease. As nine cases showed a minor degree of prothrombin deficiency even though they were on good convalescent diet, it is possible that the marked fall in the two cases is due to toxicity and dietetic faults acting side by side. But, in no case did we get prothrombin deficiency to such a marked degree as to serve as an index for bleeding tendency. Yet some of the patients in the group of cases we tested were getting hæmoptysis now and then and one of them had an attack a few days after the test, though his prothrombin time was normal. From our results we cannot conclude that prothrombin deficiency is the real cause of hæmoptysis in tuberculosis, though a certain fall in blood prothrombin may be present in some cases of tuberculosis.

Summary

1. Tuberculosis patients who had hæmoptysis and were treated with hemoplastin, congo red, calcium, etc., did not show definite prolongation of prothrombin time.

2. Tuberculosis patients with definite hæmoptysis but who had no such treatment, did not show prolongation of prothrombin time.

3. Patients without hæmoptysis, undergoing A. P. treatment showed a prothrombin time either equal to or slightly shorter than the control.

4. Patients without hæmoptysis and without A. P. treatment also gave a normal prothrombin time, irrespective of their diet.

5. Our conclusion is that hæmoptysis in tuberculosis patients is not due to a reduction in the plasma prothrombin.

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N. B.—After the completion of this paper, we read an article on "Prothrombin deficiency in pulmonary tuberculosis" by R. F. Sheely (*J. A. M. A.*, 1941:117:1603). The investigation was started to determine whether there occurred any disturbance in the mechanism of coagulation in patients suffering from acute or chronic forms of pulmonary tuberculosis. Quantitative estimation of prothrombin was done according to Quick's method. Leaving the many details and figures, the observations and conclusions of the author may be summarised under two headings: (1) Relation of the level of the prothrombin to tubercular infection. Out of the 106 cases studied, 51 cases showed significant deficiency. Among 13 cases of hæmoptysis, 8 had prothrombin concentration of 45 to 35 per cent. "There appears to be a relation between the prothrombin concentration in blood and the degree of toxæmia as measured by clinical standards in pulmonary tuberculosis." The relation between the extent of the tubercular involvement and the prothrombin level was not so certain. There was an elevation of prothrombin level parallel to the improvement in clinical condition. In chronic inactive tuberculosis, regardless of original extent, prothrombin concentration was markedly within normal range. It is, therefore, suggested that prothrombin concentration should be done as a routine test in all cases of hæmoptysis. (2) Effect of vitamin K on hæmoptysis. Four cases of hæmoptysis improved with the administration of synthetic vitamin K. It is pointed out that the new drug may be an addition to the armamentarium in the control of hæmoptysis in certain cases of pulmonary tuberculosis.

ACUTE INTESTINAL OBSTRUCTION : DIAGNOSIS AND TREATMENT*

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The management of acute intestinal obstruction is always a serious problem and more so in this country as the majority of cases come very late—in our experience, on the third or fourth day—and they have usually had various treatments applied in the village which have made matters much worse. Strong cathartics which increase the peristalsis and pain, and also the vomiting; massage of the abdomen which damages the distended intestine; and blistering of the skin, which makes operation more dangerous—all these contribute to the high mortality.

Yet, it is not only under conditions such as these that the mortality of acute intestinal obstruction is high. Even in countries where doctors and hospitals are more numerous, and where the people are more accustomed to seek trained medical aid earlier in the course of the disease, the percentage of recoveries from acute intestinal obstruction has been all too small—certainly until very recent years. There as well as here, too many of the patients come only after taking a cathartic, unfortunately sometimes given by the first physician who sees the case. It has taken a long time for doctors, and, of course, will take longer for patients, to learn the value of the dictum: For pain in the abdomen a cathartic should not be used; if the pain is accompanied by constipation an enema may be used instead as a diagnostic procedure.

However, even though the patient presents himself within the first 24 hours of the onset of obstruction, there is sometimes a delay on the part of the physician for there is often a real difficulty in arriving at a correct diagnosis in the early stage of intestinal obstruction. To recognize that intestinal obstruction is present is not difficult in the late cases, but even in these the diagnosis as to the type of obstruction and its cause is most important for the treatment and the prognosis vary a great deal depending on the type of obstruction present.

Because of the importance of accurate diagnosis in the handling of intestinal obstruction, and especially because of its importance in selecting certain cases and rejecting others for the use of the Wangensteen apparatus, we should like to consider certain important points concerning diagnosis before proceeding to the question of treatment.

While the presence of the classical symptoms: abdominal pain, obstipation, distension and usually vomiting point to a diagnosis of acute intestinal obstruction, one must make a detailed analysis of these and the other symptoms and signs, and also consider the patient as a whole and the question of relative frequency of occurrence in arriving at a correct diagnosis of the type and location of the obstruction.

McIver (1934) gives the incidence of various types of obstruction in 335 cases from the Massachusetts General Hospital as under:

Strangulated external hernias 44 per cent, bands and adhesions (postoperative) 24 per cent, bands and adhesions with previous operation 6 per cent, neoplasm 10 per cent, intussusception 5 per cent, volvulus 4 per cent, mesenteric thrombosis 3 per cent, gall stones and other foreign bodies 2 per cent, internal hernias 0·9 per cent, Meckel's diverticulum 0·6 per cent and congenital anomalies 0·6 per cent.

Since strangulated external hernia is by far the most common, one must look carefully for small epigastric, umbilical, or femoral hernias as well as for the more obvious inguinal variety in all cases of intestinal obstruction. In some cases in which a definite hernia cannot be found by physical examination, the history may be suggestive. Two recent cases illustrate this point. A man gave the history of a mass going down from the right inguinal region and getting stuck for a day or two in the scrotum, several years before. This time he had slight pain in the right inguinal region following straining at stool, but there was no palpable mass. He was obstructed from then until admission. The intestine, but not the mesentery, was obstructed in the internal ring. A woman in whom we found about one inch of intestine stuck in a hole in the left obturator

* The paper is in particular reference to experiences with the Wangensteen Duodenal Suction Apparatus.

foramen gave a clue from the fact that she complained of an indefinite pain in the left thigh.

In addition to considering the probabilities of different types of obstruction based on such statistics of a large series of cases, one should consider further the fact that in certain types of patient the incidence is higher for some kinds of obstruction. In infants and children the predominant cause is intussusception—88 of 105 cases reported by Ladd and Cutler (1924), and 46 of 55 cases reported by Peterson (1922). In elderly patients, cancer of the colon and volvulus of the sigmoid are more common. In patients showing a scar of an abdominal operation and in any patient who gives a history of previous inflammatory disease in the abdomen, bands and adhesions are a common cause of obstruction. When obstruction occurs soon after operation, the element of inflammation or of soft adhesions is more common, and non-operative management may more safely be tried, but in late postoperative obstruction (over 4 weeks and sometimes as long as 25 years after operation), Mc Iver (1934) reports a high proportion of cases with strangulation (20 out of 45 cases).

There is probably no condition in which it is more worth while to spend sufficient time in taking a careful and detailed history and in doing a detailed physical examination. We have found it very valuable to refer frequently to the excellent monograph on Intestinal Obstruction by Mc Iver, who points out the significant points to be sought in history and physical examination.

Lewis, Shapiro and Vaughan (1940) emphasize the importance of auscultation of the abdomen as well as the use of stomach tube, liquid paraffin, x-rays and 3-pint enema in the diagnosis of intestinal obstruction. They also bring out a number of significant points:

1. Many cases of mechanical obstruction are incomplete. 2. In these cases there is no fever, none or little tenderness, but marked cramplike pain accompanying the increased peristalsis and marked increase in borborygmus. 3. When obstruction has become complete and in most of the cases of strangulation, the abdomen is silent, except for a high pitched sound on succussion, while tenderness is marked and fever is usually present.

The patient who has waited at home for 2 to 4 days is nevertheless anxious to get immediate relief when he comes to the hospital. But a hasty diagnosis and an immediate laparotomy are not to be recommended as the best response to the patient's urgency. We have heard that our veterinary colleagues can cure the bloated abdomens of certain animals by simply poking a few holes into the dilated intestines through the belly wall. A somewhat more refined but similar technique was forced on the surgeon who used to open the distended human abdomen. Yards of distended intestine popped out and an ileostomy for deflation had to be done just to close the abdomen—often without any chance to find the cause of the obstruction.

This situation has been changed completely by the coming of the method of internal deflation by duodenal tube suction. We can do something immediately to relieve the patient, and while his stomach and intestine are being deflated, we can give intravenous fluid and take time to make a diagnosis as to the cause of the obstruction. And during this time the patient becomes a better risk for operation if such is required.

There is adequate experimental basis for the use of internal suction before operation. Sperling (1938) has shown the vicious circle of distension and Scott (1938) that distension rather than toxic absorption is the important factor until the final stages of the condition.

As Coller (1938) points out it is well for us who are constantly busy with what we call practical clinical work to recognize the value of the laboratory contribution to the diagnosis and especially the treatment of intestinal obstruction. The laboratory observations of Hartwell and Hoguet were the background for Haden and Orr's introduction of intravenous saline in adequate amounts in the treatment of intestinal obstruction. And the Miller-Abott tube used for physiological research was adapted to be used with Wangensteen apparatus.

For a number of years we have been using intravenous saline as a preparation for operation in intestinal obstruction. In some of their early work, Haden and Orr advised the use of hypertonic (3 per cent) saline, but they and other workers have since returned to the use of isotonic saline often with the addition of glucose. We now give several pints of saline and glucose to each patient soon after admission, the amount depending on the degree of dehydration present.

However, it is only since we started using the Wangensteen apparatus that we have met with any measure of success in dealing with intestinal obstruction.

The prompt relief of distension of the stomach stops vomiting. Gradually in many cases the small intestine is also deflated and the operation is easier to perform and the closure of the abdomen tremendously simplified.

At first we used the apparatus strictly as a preoperative treatment. Later we had a patient in whom the symptoms of obstruction were entirely relieved and he began passing gas and then feces per rectum. This change was so spectacular that we continued to use the suction, and then began to give nourishment while we clamped off the suction tube. In that case we should have operated while the patient's condition was better, for he again became obstructed, and later died following an operation in which we found a band which caused obstruction in the ileum which had apparently been partial at first but later had become complete.

Our later experience with the apparatus and some improvement in our diagnostic ability has shown us more definitely what are the indications for and the limitations of this very valuable therapeutic procedure.

Briefly described, a simple Wangensteen outfit is a duodenal tube which can be introduced through the nose, to which is attached a simple system for producing moderate suction. Siphonage is not sufficient because of the presence of gas as well as fluid in the stomach and intestine. The amount of gas removed is shown in the upper bottle, and the amount of fluid in the lower bottle.

The following are some of the cases in which we have used this apparatus. The cases are selected to illustrate certain differing types of obstruction, as well as to point out the place which the duodenal suction technique occupied in the course of treatment.

Case 1—Man aged 45 with severe pain, marked distension, extreme dehydration, with a temperature of 97·6° and pulse of 104, and vomiting bilious fluid. History of an attack of similar cramp-like pain and obstipation 7 years ago. Present illness started 6 days ago. Temporary relief with enemas in another hospital. He refused operation and went to his village. He became worse, so was brought to our hospital. *Treatment*—Enema given. No result. Wangensteen apparatus connected continuously for two days. On the third day distension was completely gone. Saline and glucose were given intravenously and rectally during these 3 days and later by mouth; Patient discharged apparently well. We advised exploratory laparotomy which he refused. We think this was a case of incomplete mechanical obstruction which may recur.

Case 2—Man aged 50, was admitted for abdominal pain, vomiting, distension of abdomen and constipation for at least 2 days. Condition on admission—The patient was in shock, dehydrated, in agony. *Treatment*—Intravenous saline and glucose. Enema—no result. Wangensteen apparatus connected. Operation—Laparotomy on the next day after the shock and distension were relieved to some extent. Volvulus of the terminal ileum found. There were some hæmorrhagic spots behind the volvulus. The volvulus undone and abdomen closed up. The Wangensteen apparatus was kept working during and after operation for one day until most of the distension was gone and the bowels started working. More intravenous saline and glucose, rectal saline and frequent passing of rectal tube.

This was a case, which, though not strangulated, would have been fatal without operation. However, the preoperative preparation with deflation and hydration changed the picture from one of shock to a condition in which a successful operation could be performed.

Case 3—Man aged 33, was admitted with attacks of severe abdominal pain coming every two or three minutes, which had started suddenly the previous day. Vomiting occurred throughout that day, then stopped until after admission. The pain progressively increased and the abdomen was getting distended. He had not had a motion after the pain started despite a mild native purgative and an enema. The lower abdomen was dull and loops of intestine were easily made out. He was quite dehydrated. *Treatment*—We gave him some saline and glucose intravenously and applied the Wangensteen apparatus. The distension diminished. We did a laparotomy with the apparatus *in situ*. We found strangulation of small intestine by a band from a Meckel's diverticulum. The strangulated bowel was gangrenous. It was resected and lateral anastomosis was done. The suction was continued for three days after operation. The patient made an uneventful recovery.

Case 4—Man aged 21, was admitted with the history of numerous watery motions for three days: Vomiting and cramp-like pain for one day. He was in shock and looked very much like a cholera patient. *Treatment*—During the first two days glucose and saline were administered intravenously and rectally. The Wangensteen apparatus was connected on the third day when distension was noticed to be increasing. Distension was relieved but whenever the apparatus was disconnected and nourishment given by mouth, pain and vomiting occurred. So exploratory operation was done, which revealed the following conditions: distended small intestine, bluish but not gangrenous; the last portion of the ileum was collapsed and the lower end of the cæcum including the ileocæcal junction was found twisted up under the upper portion of the cæcum; the appendix, the end of which was kinked, was fastened up to the side wall of the lumbar gutter by a band; and another band (apparently the normal anchoring liganent) was pulling downwards on the cæcum toward the iliac fossa. When these bands had been cut, gas and fluid went into the cæcum and up into the transverse colon. Then appendicectomy was done, with the idea of preventing the recurrence of the trouble.

The next day after operation, the patient was suddenly found to be in shock and despite stimulants and artificial respiration, he died.

This case illustrates that in some cases of obstruction especially those in the lower ileum, in which the development

of symptoms is naturally slow, there may be a danger in the very relief which the Wangensteen apparatus gives, because the development of the full picture of complete obstruction is delayed and masked. It was a case of partial obstruction of the band type at first, and gradually developed into complete obstruction. Though the retrocæcal appendix, and the twisting of the cæcum and ileocæcal junction caused by it were the original cause of the trouble we were probably at fault in removing the appendix at that operation. The severing of the bands might have sufficed.

The Wangensteen apparatus will deflate the small intestine but not the colon. In colonic obstruction there is very marked distension, but without vomiting, and only moderate dehydration and collapse considering the length of the illness. The Wangensteen apparatus will not relieve the distension in such cases. The technique of liquid paraffin and repeated large enemas is well worth trying, and may be life-saving in certain cases of colonic obstruction.

Comment

Following on the development of the Wangensteen apparatus there has been a great stimulus to the study of intestinal obstruction, both in the laboratory and clinic. And the Wangansteen apparatus has changed our outlook of the treatment of intestinal obstruction. Nevertheless, all of our problems have not been solved, and there is still great need for judgment in the matter of when to operate and still more judgment to decide when operation is not required.

The advice of Wilbur (1938) though given in a discussion of the Miller-Abbott tube method, is probably still more applicable with regard to the use of the Wangensteen method, using the shorter duodenal tube: "There are dangers in the use of this method and I should like to emphasize that when suction is applied and the intestinal contents are drained for any period the situation is comparable to that in which an enterostomy opening is present". (We have been interested to note that the reports of the use of this operation are almost absent since the appearance of the suction methods). "Under such circumstances considerable care must be exercised to maintain an adequate balance of fluid and chloride. There is danger also in the patients who have strangulation of the small bowel, and the possibility that strangulation exists will probably constitute the greatest limitation of this particular method of treatment of intestinal obstruction. The principal therapeutic uses of the tube probably will be in the treatment of obstruction of the small intestine which is mechanical in nature, in the treatment of paralytic ileus and in the preoperative treatment of patients with obstruction who have marked abdominal distension. I think it will be wise for those who have not had much experience with this method of treatment to be prepared to operate in the first few cases in which it is used or until such time as familiarity with its technic and with its possible limitations has been developed". However, the apparatus is of value even for isolated medical officers who do not operate. The patient who is deflated before a journey to the larger hospital is a better surgical risk.

In conclusion, another word should be said about the Wangensteen apparatus. It will not work itself. In fact it requires rather close observation by the doctor and nurse, especially during the first few hours after the tube has been introduced. Thereafter it needs to be inspected rather frequently and possibly adjusted, or tested to see whether the suction is working. But with careful attention, we have found it to be a very valuable addition to our armamentarium for the handling of cases of intestinal obstruction. It also has been found useful in treating nonobstructive distension, post-operative and postpartum, and in medical cases *e.g.*, pneumonia.

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TREATMENT OF PELVIC DISPROPORTION BY CAESARIAN SECTION AND INDUCTION OF PREMATURE LABOUR

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It has been recognised that in Southern India, female pelvis is comparatively smaller in size than what is noticed in the northern provinces. While 9½" intraspinous and 10½" intracristal diameters are taken as standard for normal female pelvis, we in Hyderabad quite often meet women having 8½" and 9½" as corresponding measurements. Similarly, 7½" is considered normal external conjugate for a good size pelvis, but here we have delivered a normal healthy child through 6½" external conjugate. Thus arises the popular conception that the head of the fœtus is a proper guide rather than the pelvimetry commonly employed. I have collected a record of 26 cases of rickety flat pelvis who have undergone the operation of cæsarean section. The number is rather small for proper scientific investigation, yet one can learn a good deal from these observations and results.

These cases fall in three main groups:—(*i*) 18 cases of primipara with definite contracted pelvis. (*ii*) 3 cases of those who have undergone cæsarian section previously at least once in life. (*iii*) 5 cases of relative sterility *i.e.*, those who each time deliver still-born fœtus or had difficult forceps extraction resulting in the death of the fœtus or craniotomy performed, ultimately yielded to cæsarean section and live child delivered.

I may also point out that most of the recorded cases were operated and delivered within twenty four hours of their admission to the maternity ward and thus deprived from antenatal investigation, which is becoming all important in the teaching of modern obstetrics. Nevertheless for the sake of hospital record, we have taken on admission pelvic measurements as carefully as possible but mostly relied on the disproportion of the fœtal head to the pelvis in question during the progress of the labour.

The clinical records of these 26 cases were briefly as follows:

1. Twenty cases were admitted with floating fœtal head and delivered within 24 hours by cæsarean section, after undergoing a fair trial of labour.

2. Five cases were admitted a week or less prior to onset of labour and delivered by cæsarian section, after having short trial of labour.

3. One case was conducted by internal podalic version but baby died due to labour trauma.

In this series we had luckily one maternal death due to puerperal septicæmia and one fœtal death mentioned above.

Almost all these cases were found to have rickety flat pelvis and I have purposely excluded other types of contracted pelvis as it will unnecessarily lengthen the discussion. Our method of investigating the disproportion of the fœtal head to a particular pelvis has been on the line of Muller and Munro Kerr whose methods are well-known. Those methods were applied before and during labour in order to divide cases into three main groups, namely:

1. Those on border line or with 1st degree contraction of the pelvis with probability of delivery per natural passage.

2. Doubtful cases with 2nd degree contraction of the pelvis.

3. Cases with marked disproportion falling in the category of 3rd degree contraction of the pelvis. Thus we endeavour to find whether the head can go through the brim of the pelvis to the level of posterior ischial spine, if so it is safely left for the natural delivery, otherwise, if the fœtal head just enters the brim and may not be pushed through it is labelled as border line case.

Another type is where fœtal head just bites the brim and is usually flush with the symphysis pubis. I call it a doubtful case for delivery per natural passages.

Lastly, there are cases where the fœtal head is overriding the symphysis pubis and will never go through the brim at any stage of the labour.

Thus, if we keep these four points in view while examining a case of pelvic disproportion our decision for treatment becomes rather easy.

When the head is overriding the front of the symphysis pubis, it is a clear-cut case for cæsarean section; premature induction of labour and trial labour are both equally risky as they might lead to rupture of the uterus.

But when the fœtal head is able to enter the brim and is not through the brim or as in 2nd degree flush with the symphysis pubis—premature induction of labour coupled with trial of labour may carry the fœtal head through the relatively narrow pelvis.

We have not tried premature induction of labour on a sufficient number of cases to pronounce a definite result; as already stated, most of these cases were delivered within twenty four hours of their admission in the hospital. But my experience on private cases and what I have observed in certain well-known British and Irish hospitals as well as here confirms the opinion that in selected cases such as falling in the 1st and second degree of pelvic disproportion, premature induction of labour has got definite place. I have decidedly observed good results from induction of premature labour in cases where head begins to arrest at the brim and the term of pregnancy has not exceeded 38 weeks. Such a case has the advantage of having small soft and easily moulding fœtal head, thus capable of overcoming relatively slight disproportion and ending into spontaneous delivery, while the head as I described just bites the brim of pelvis and flush with the symphysis pubis and with conjugate vera 3½" or below; the prognosis for vaginal delivery is doubtful. But here again induction of premature labour combined with trial is worth while in order to find out what can be done.

As regards the management of trial labour, I do not wish to indulge in the detail of the method employed, but a few points must be remembered with the view of knowing its progress or failure otherwise. In our cases the time allowed for a trial labour extended from 6 to 18 hours at average, in a few cases longer time was allowed as pains were weak for the most of the time.

For the success of trial labour we take several factors into account. The condition of the mother should be satisfactory to endure the long strain of labour, fœtal heart must not exceed 145 per minute, good strong labour pains are well-established and maintained for over two hours or more and finally whether the head is descending and engaging into the pelvis having maintained complete flexion. Vaginal and preferably rectal examination should be carried out at reasonable intervals to find out the dilatation of the cervix.

Signs of failure of the trial labour are distinct and few which must not be overlooked. If the cervix does not continue to dilate inspite of having strong labour pains extending over couple of hours or has actually reformed again and not applied well to the fœtal head, prognosis is bad.

If the head remains extended and floats above the brim of the pelvis inspite of good labour pains the moulding of the fœtal head will be incomplete and the constant pressure over the head from above (due to contraction of uterus) may lead to anterior or posterior asyncliticism of the head and ear presentation. Under such circumstances test of labour has completely failed and recourse to cæsarean section remained opened.

In conclusion, I wish to draw your attention that while cæsarean section is an operation of choice in good many cases of contracted pelvis with conjugate vera below 3½". Induction of premature labour has got a definite place in selected cases.

Circumference of fœtal head plays an important part in its easy passage through the brim of the pelvis. A male fœtus has usually larger head than a female child though this is not true always. Therefore, it is possible that a female fœtus while passes through the same pelvis the head of the male child may arrest at the brim. This phenomenon I have observed in one of my cases with flat pelvis who delivered a female child as primipara as breech and at second delivery the head of the male child also a breech case arrested at the brim and removed by craniotomy.

A trial of labour is worth while in cases with 7" to 6¼" conjugate vera.

In a tropical country like India, 38 weeks of pregnancy is suitable time for the induction of premature labour in view of under-weight fœtuses born in this country.

Antenatal care and examination by Munro Kerr and Muller's method at regular intervals of pelvic disproportion cases are deciding factors in the outline of treatment.

Fœtal head is a better guide than the pelvimetry in deciding the passage of the head through a particular pelvis.*

* Read at the Scientific Section of the XVIII All-India Medical Conference held at Hyderabad-Deccan, December, 1941.

GASTROGENOUS DIARRHOEA

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Introduction

Gastrogenous diarrhœa may be defined as a form of looseness of the bowels seen in conditions of gastric subacidity and anacidity. The main symptoms in the twenty five cases studied were: diarrhœa, lack or loss of appetite, soreness of mouth with or without glossitis weakness and wasting, occasional flatulence. Colicky pain was only of rare occurrence.

Of these, the most outstanding is the diarrhœa. The characteristic feature of the diarrhœa associated with hypo- and anacidity is the fact that looseness of the bowels is provoked by a meal or may follow a short time afterwards.

Two types of cases are thus met with: (*a*) Those in which motions appear almost immediately after the food is introduced into the stomach, when the motions may be frequent, yellowish, foamy and even fœtid in character. (*b*) Those in which movements appear ½ hour or more after ingestion of the meal when the first motion is soft and almost normal while the subsequent ones are liquid. Most of my cases belonged to the first group. There was hardly any pain in any of the cases.

Clinical Features

Motions—Naked eye appearance:—There were no mucus, pus or blood. Connective tissue, vegetable remains and froth were present. Reaction—Usually alkaline. This I consider to be important from the point of view of diagnosis. Microscopical examination—Entamœba coli, trichomonas intestinalis, and Lamblia are commonly present, the last named being the most frequent in the cases studied.

Lack or loss of appetite—This symptom is very important in diagnosis and specially helpful in differentiating it from sprue in which the appetite is apt to be capricious and the patient may be very obstinate and indulging on dietary indiscretions.

Soreness of the mouth with or without glossitis—There may be just reddening and excoriation of the mucous membrane or vesicles may appear and break down to form shallow ulcers. In extreme cases the tongue may be quite bald, red and angry-looking, resembling that obtained in pellagra and becomes coated again in due course after oral administration of nicotinic acid. From the point of view of clinical diagnosis this sign in tongue is of great significance so much so that there will be little hesitation in diagnosing hypochlorhydria or achlorhydria in a patient who complains of diarrhœa, loss of appetite, soreness of mouth and whose tongue is bald. The order of appearance of the symptoms is also helpful in clinical diagnosis. Thus in sprue the first symptom is soreness of the mouth and diarrhœa follows it whereas in gastrogenous diarrhœa the first symptom is loss of appetite followed by diarrhœa, while soreness of the mouth comes last. If this point is kept in view gastrogenous diarrhœa will not be mistaken for cases of sprue.

Weakness—A patient who has been a victim of loss of appetite and diarrhœa along with soreness of the mouth is unable to take food and naturally feels weak and exhausted and necessarily loses weight.

Flatulence—Colic was only rare in my cases in contrast to the description of gastrogenous diarrhœa given in books which mention colic, flatulence and diarrhœa as the cardinal clinical features. In my opinion this is a thoroughly accurate picture of carbohydrate dyspepsia and one of my cases exhibited these symptoms. Practically none of my cases of gastrogenous diarrhœa complained of pain and this is in exact accordance with the findings of Napier and others who state that cases with high acidity are associated with epigastric pain, and those with low acidity with diarrhœa. Anæmia is not an essential component of the picture in early stages but later on as the disease advances and malnutrition supervenes, anæmia sets in and œdema appears round the ankles.

Diagnosis

The diagnosis of the condition is easy. If a patient complains of loss of apetite, diarrhœa, soreness of mouth and weakness with or without flatulence or pain, the gastric acidity must be investigated.

The condition, however, must be differentiated from: (*i*) Sprue (stool—pale, pasty, fermenting and acid); (*ii*) Pellagra (of the three D's of pellagra, dementia, dermatitis and diarrhœa there is only one D of diarrhœa here, no dermatitis and no dementia); (*iii*) Lienteric diarrhœa; (*iv*) Tropical megalocytic anæmia; (*v*) Pernicious anæmia; and (*vi*) Pancreatic disease.

Etiology

Napier and others have studied the gastric acidity of various communities in Calcutta and have found that the gastric acidity is not lower than European standards. My findings have been that the gastric acidity in vegetarians is slightly lower than that in nonvegetarians and that in nonvegetarians it is slightly lower than in Europeans.

Etiological factors that may bring about hypoacidity or anacidity may be enumerated as follows: (1) Constitutional factor. It is stated that 4 per cent of healthy individuals at the age of 20 and 10 per cent at the age of 40 have hypochlorhydria—a familiar condition occurring equally in males and females. (2) As a result of treatment like partial gastrectomy. (3) Allergic conditions *e.g.*, asthma, skin irritants and protein sensitizations. (4) Temporary gastritis which give rise to achlorhydria is generally the sequel of prolonged irritation which may be: (*a*) mechanical due to (*i*) excess of coarse vegetable material including roughage specially in vegetarians, (*ii*) insufficient mastication, (*iii*) deficient dentures. (*b*) chemical irritants—excess of curries, pickles, chillies, condiments and spiced chutneys, sendi or toddy taken on empty stomach, strong tea and coffee, excess of lime and nuts; (*c*) bacterial factor in the form of oral sepsis. Some oral sepsis was present in nearly every case I studied.

Temporary gastritis acts in the following ways: (*i*) inflammation of the gastric mucous membrane impairs the functional activity of the damaged secreting cells; (*ii*) The swollen cells obstruct the intra- and intercellular canaliculi which carry the acid from oxyntic cells to tubules; (*iii*) goblet cells situated between the tubules secrete an excess of mucus to protect the mucous membrane from damage. But the mouths of tubules become blocked and secretion is prevented from gaining access to the lumen of the stomach.

Treatment

(*i*) Rest in bed. (*ii*) Cut down all the irritants in the food. Miik, sago, plain meat soup, strained fruit juice, egg flip are the best. The first thing in the morning while fasting the patient should take soda bicarb grs. 60 dissolved in soda water one tumblerful. This would wash off the mucus from the surface of gastric mucous membrane and soda bicarb is the strongest of all stimulants of gastric secretion. If this does not restore gastric secretion, the gastric lavage is tried every morning when the patient is fasting with hydrogen peroxide 1 dram to one pint of water gradually increasing to 4 drams to pint of water till the quantity of mucus diminishes. It should be continued with successive quantities of about 5 per cent until the washings are quite clear. The final lavage should then be made with plain water. The treatment should be continued without interruption until the first washing is quite clear and it is repeated on alternate days for a week. At this stage test meal is done again and if alchlorhydria is still present begin dilute hydrochloric acid by mouth in the form of a beverage during lunch and dinner. (*iii*) Dilute hydrochloric acid 15 to 30 minims with meals. Prevent erosion of the teeth by dilute hydrochloric acid. (*a*) provide two glasses at meals one containing acid drink, the other plain water. Patient should drink a little plain water after each mouthful of acid drink in order to wash it away from the teeth or (*b*) diluted acid should be taken through a glass tube to avoid coming into contact with the teeth. (*c*) Immediately after each meal the mouth should be thoroughly washed with water containing soda bicarb and mag. hydroxide. The return to the ordinary food should be very gradual.

Summary and Conclusion

Gastrogenous diarrhœa was seen in persons having gastric hypoacidity or anacidity. The present paper is based on 25 cases.

The clinical picture of gastrogenous diarrhœa along with its etiology, pathology, diagnosis, differential diagnosis and treatment is discussed.

Gastrogenous diarrhœa is a definite syndrome quite distinct from sprue with which it may be easily confused and is not so rare as mentioned in text books.*

* Read at the Scientific Section of the XVIII All-India Medical Conference held at Hyderabad-Deccan, December, 1941.

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WANTED DOCTORS

India is a sub-continent. It is as big as Europe without Soviet Russia. The population as found in the last census is about 390 million. It shows an increase of about 15 per cent during the last ten years. The question is: have we enough doctors to render scientific medical aid to this population. From a communique of the Hon'ble Minister of Public Health with the Government of India, we find that the total number of medical men including all graduates and licentiates is 42,000. Calculating on this figure we have 1 doctor for every 10,000 persons. A modern society has a right to expect at least one physician for every 1,000 inhabitants. The ratio is thus hopelessly meagre when compared with other civilised countries, although the first Medical College in India was established more than 100 years ago. The result is that the suffering population becomes the prey of quacks. On the other hand, our country has been the play ground of plague, small-pox, malaria, kala-azar, cholera, typhoid, dysentery and other communicable diseases for the last century. While other countries of the West have been made free from the ravages of these diseases during the last 60 years, in India there has been no change. There are still 6·1 million annual mortality from these diseases. The general expectancy of life is 23 years. To add to this misery the average income per head is about 2½d. a day. The general resistance is low. Deficiency diseases are on the increase. Even in this 20th century there is no scientific method of recording the death rates in the rural areas. Such a condition coupled with the static public health measures makes a strong argument for more qualified men.

Now-a-days doctors are mainly distributed in the urban areas. Like all human beings, they want to earn their living and for this reason the rural areas offer no attraction for them. The rural population cannot afford a doctor or medicine for his ailments because they have to struggle more for one meal a day. The greater part of the population suffer and die untreated and uncared for. Calling in a doctor is a luxury which very few can enjoy in the rural areas. Under these circumstances, our colleagues have done and are doing their best. But as India does not manufacture most of the medicines, it becomes absolutely impossible to give medicines for diseases, unless from one's own pocket. Doctors cannot raise the economic level of the people or produce drugs at a price which the people can buy. As a solution many people are talking of a Health Insurance Scheme in rural areas. How can a Health Insurance Scheme function with a daily income of 2½d. without adequate State aid? It is an impossible proposition or a mere wishful thinking or a lack of appreciation of the real condition of the population unless the economic level is raised at least 14 times.

With the congestion of doctors in the urban areas, unemployment among them has increased. When millions of people are dying in this country every year from preventable and curable diseases, unemployment among the medical profession seems a tragedy. The whole structure of Public Health administration in this country is based on a scrappy and unimaginative idea. Otherwise, why this anachronism? Reactionary elements suggest admission of lesser number of students in medical colleges and schools or closing down some of them. If there are less doctors, there is less competition and division of income. As a result, unemployment among doctors will be solved. Can anything be more suicidal to the nation? Are we to go back to mediævalism and barbarism and laws of the jungle after 50 years or so, when disease and destitution will run rampant in this country? Shall we turn back instead of looking forward?

In spite of the low economic level, lack of nourishment and ravages of disease, the population of our country has increased. Isn't this a fair ground for optimism. Scientific agriculture, industrialisation, reclamation of waste land, fertilisation, irrigation and better legislation for the land should start immediately after the war. Doctors should be given more encouragement, not in mere words of serving humanity, to go to the rural areas. Their income should be secured by State services. Village hospitals should be started and equipped by the government.

Once we have started, there is no going back. Reactionary ideas of less students in schools and colleges should be banished once and for all. The country with its suffering population wants treatment and eradication of the vile diseases. This problem can only be solved by more doctors and their distribution throughout the country with a little imagination and sympathy.

WAR AND MORALE

The entrance of Japan has marked a new era in the history of the present war. So long we had been, at best, sympathetic listeners to the devastations of war and the serious hardships to which the civil population has been subject not only in the absolute war zones but also in areas far remote, due to air attacks with high-explosive or incendiary bombing or even machine-gunning; but there was no immediate danger of direct attack on India.

A new feature of the present war, however, is the air attack of important towns—not only on military objectives but also on civil population partly by mistake and partly on principle to break the morale of the pepole to make normal life out of gear and thus make difficult the supply of food and ammunition to the army.

Though the biting fangs of military machinations of the enemy have not yet come directly on our heads, its indirect havocs are already felt in the various social spheres of the country—the prices of essential commodities have already risen too high and are still rising by leaps and bounds day after day; the teaching staff in schools and colleges are out of employment; the professional people have lost their support; many business have been wound up, so many employees have to leave; students are out of their study due to evacuation; many peaceful families have been separated for the same reason; and last but not the least various diseases are making their appearance in the villages, which again have become uninhabitable being over-crowded in the absence of public health measures, properly trained doctors and even everyday medicines.

It is not difficult, therefore, to understand the minds of individuals who, not being allowed to leave the cities with their families would be at the limits of tolerance and their mental stability reserve completely used up. Some of them at least will break down and get various mental disorders which may, therefore, appear in an epidemic form on the first bombing outrage; moreover, they may in their extreme panic and anxiety, elaborate and circulate rumours and even infect others and create mental breakdown in the most stable individuals.

The best arrangement for the prevention of such a catastrophe is either to examine every ordinary and essential individual beforehand as regards their mental reserve or the arrangement of immediate treatment for those who get mental breakdown. As the previous examination of all individuals is not feasible, arrangements should be made in every first class A. R. P. Hospital of all the big cities in India for the immediate psychological treatment of anxiety states, bombshock, nervous exhaustion and so forth.

No elaborate arrangement is necessary. Doctors and psychologists were mobilised to meet these emergencies in England. But many of their expectations were not realised. Big hospitals set up to deal with nervous cases were not required. After three years of terrible warfare the number of so-called war neuroses is not greater than that in the last war. It is said that this has been due to the sense of humour, preparation for self defence, public morale, *esprit de corps* and *sang froid* of the English men. We believe these will not be lacking in India when occasions arise. While arrangements for war neuroses are made in different hospitals the public must be educated to practise self knowledge, self discipline and to put up a brave front. Reality experiences will be less terrifying than phantasy ones. Now-a-days there is a certain amount to be said in favour of living dangerously and adventurously. Here are a few principles of what has been aptly termed Mental First Aid[1]:—

1. Practise self knowledge; come to terms with your fears. Admit your fears, learn to recognise your unreal and your exaggerated fears and learn to laugh at them.
2. Practise self discipline; make a cool appraisal of real danger and be prepared for the worst that can happen.
3. Be prepared for action; because effective action drives away fear.
4. Put on a brave front; it helps yourself and a good example is infectious. But do not try to be a hero; just try to be yourself.
5. Practise mutual aid; be neighbourly and encourage community feeling. Loneliness increases fear, so be social.

MASS PREVENTIVE INOCULATION

From the experience of World War I, we know that epidemic diseases break out in a virulent form and cause wide spread devastation immediately after the cessation of hostilities. It has been estimated that the total number of mortality exceeded the number of casualties in actual warfare. War is now at the door of India. In peace time the mortality from communicable diseases like cholera, typhoid, small pox and plague is very high. Immediate measures should be taken to inoculate the public against such preventable diseases. Arrangements for free inoculations should be made in every town and village. We draw the attention of the Public Health Authorities and the medical profession to this important 'Nation Saving' measure.

1. Bowley, Agatha. H. (1942)—The Natural Development of the Child. Published by E. & S. Livingstone, Edin., p. 153.

CASE NOTES

A CASE OF MALTA FEVER FROM CAWNPORE

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The patient, aged 38, started getting fever in the month of January, 1941. It was diagnosed a case of typhoid fever by local doctors and was treated on those lines. His temperature came down after 2 months and remained normal for 15 days. He again began to keep temperature which rose up to 101°F. in the evening. He was now suspected to be a case of pulmonary tuberculosis inspite of the absence of cough and definite physical signs in the lungs. He was sent to a hill sanatorium where he remained for 3 months. He got better and his skiagram of the chest gave no indication of tuberculous condition. When he came back to Cawnpore he again started getting temperature. Except for the definite enlargement of spleen and liver (2 fingers below the costal margin) he showed no positive physical signs. Malaria and kalazar were considered, but, his formaldehyde and urea-stibamine tests were completely negative. His blood count was as follows: Total W.B.C. 6000 per c.mm. Differential count: polymorphs 55 per cent, lymphocytes 42 per cent, large mono 3 per cent. No malaria parasites were detected. The result of agglutination test against the organisms of typhosum group [B. typhosum (H, O and Vi); B. Paratyphosum A, B & C—(H) and Group Salmonella (H)] was negative in dilutions of 1 in 25, 1 in 50, 1 in 125 and 1 in 250.

As all these tests were negative Malta or Undulant fever was suspected. The fact that the patient was fond of goat's milk and sometimes used to take it unboiled strongly confirmed the suspicion. An agglutination test with Brucella abortus and Brucella melitensis was put up. It was found to be positive up to a dilution of 1 in 500 for Brucella melitensis. This finally clinched the diagnosis.

Malta fever is considered to be rare in this part of the country and I have seen only two cases in the last ten years. This case reminds us that malta fever should always be kept in mind in cases of prolonged fever who are usually labelled tuberculous inspite of the absence of any positive signs and symptoms.

My thanks are due to Dr. K. P. Mehrotra who referred this case to me.

A CASE OF CEREBROSPINAL MENINGITIS

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Ram Lagan, aged 20, Chamar by caste, on his way back from Assam where he stayed for about a fortnight fell sick in the train near about Katihar having had high fever with rigor, intense frontal headache and a few bouts of vomiting. He was escorted by a friend and admitted to the Hospital.

Condition on admission—Temperature 102°F, pulse 90 p.m., respiration 20 p.m., pupils dilated, Kernig's sign present, there was rigidity in the neck muscles and the abdomen was retracted. The patient was very irritable though unconscious and at times delirious. The cerebrospinal fluid was drawn out by means of a lumbar puncture—the fluid was turbid and contained pus and intracellular diplococci (meningococci).

Treatment adopted—(1) A diaphoretic mixture with 10 grains of sodium bicarbonate per dose was given thrice daily. (2) Ice cap. (3) M. & B. 693 2 tablets every 4 hours. (4) Antimeningococcus serum—20 c.c. given intramuscularly preceded by an injection of ½ c.c. of adrenaline chloride (1 in 1000) solution.

Course of the disease—The same evening the temperature came down to 101°F and was 100°F next morning when the pulse respiration rates were 80 and 20 respectively per minute. 10 c.c. of antimeningococcus serum was given intrathecally after lumbar puncture. By the evening the temperature was normal, pulse 68 and respiration 17 per minute.

On the third day the temperature recorded was 97·2°F, pulse 60 and respiration 17. The patient was constipated. In the evening the fever rose to 99·4°F.

Next day, the patient passed one good motion after a dose of a purgative and this relieved him to a great extent. His temperature came down to normal.

The patient recovered uneventfully and was discharged cured in a short time.

CURRENT MEDICAL LITERATURE

MEDICINE

Clinical Symptoms and Signs of Vitamin B Complex Deficiency

Sebrell (*Ann. Int. Med.*, 1941:15:953) writes that there are eight known substances which may be called members of the vitamin B complex and at least 4 of these appear to be needed by man and they are thiamin, riboflavin, nicotinic acid and pyridoxine. *Pyridoxine deficiency* produces symptoms of weakness and difficulty in walking and pyridoxine should be given a thorough trial in early cases of paralysis agitans. Early symptoms of *thiamin deficiency* are undue anxiety, weakness, lack of interest and a group of mental symptoms which would ordinarily be diagnosed as neurosis; the gross symptoms of extreme thiamin deficiency are enlargement of the heart, extreme œdema, tenderness of the calf muscles, weakness, hyperæsthesia and neuritis which characterize beriberi. Normal adults require about 1·8 milligrams of thiamin daily. *Nicotinic acid deficiency* producing pellagra with typical skin lesions is unmistakable but there are many cases with minimal or no skin lesion at all—they show loss of weight, weakness, mental depression, gastrointestinal upset with indigestion and diarrhœa, sometimes with stomatitis (tongue red with extensive ulceration). The normal adult requirement for nicotinic acid is about 18 milligrams daily. The symptoms of human *riboflavin deficiency* are angular stomatitis and cheilosis (the lips become scaly, appear to be chapped and are shining, reddened and denuded in appearance, specially along the line of closure) with seborrhœic dermatitis in many cases involving only the fold around the nares or the eyes, and eye symptoms such as photophobia, lacrimation, conjunctivitis and superficial inflammation of the cornea. The human riboflavin requirement is about 3 milligrams a day.

Inversion of the T Waves in Lead II Caused by a Variation in Position of the Heart

White and others (*Brit. Heart J.*, 1941:3:233) write that inversion of the T waves in lead II of the electrocardiogram, although most commonly the result of heart disease or toxic states, may be a normal physiological variation in occasional persons, particularly those of asthenic habitus with vertical hearts and prone to neurocirculatory asthenia. The position of the heart is the most important factor in producing this T wave inversion which is found in the sitting or standing position but is corrected by recumbency or by elevating the diaphragm as at full expiration. Autonomic nervous influences comprise another factor, although less striking as a rule, the low or inverted T waves then being attended by tachycardia; any cause of such stimulation, for example, excitement can then be responsible. Fear and anxiety may act through the production of overventilation with resultant alkalosis. Both heart position and nervous influences may be active in the same case. The relatively common occurrence normally of inversion of the T waves in the lead II makes it imperative to recognize its existence in order to avoid erroneous diagnosis of heart disease.

Dyspnoea

Ryle (*Guy's Hosp. Gaz.*, Ref. *Gen. Pract.*, 1942:12:324) writes that the subject may be studied (1) by obtaining a history from the patient of the type, degree and circumstances of his breathlessness and of any associated symptoms, (2) by observing for ourselves, whenever posible, the particular characters of the respiratory symptoms and signs, such as the rate and rhythm and depth of the respirations and presence or absence of the stridor, wheezing and prolongation of inspiration or expiration and investigating more closely attendant phenomena such as cough and expectoration, cyanosis or pallor and (4) by discovering physical signs—whether pulmonary, bronchial, laryngeal, cardiovascular, hæmic or nervous of the pathological condition underlying the main or presenting symptom. To systematize the studies he writes that it would not be unreasonable to classify the familiar dyspnœas into main categories based on the seat of origin or nature of an essential pathological change or disturbance—thus one can separate obstructive dyspnœas (laryngeal, bronchial or bronchiolar); pulmonary dyspnœas (pneumonic, emphysematous or fibrotic); cardiac dyspnœas (right and left ventricular); anæmic dyspnœas; central or chemical dyspnœas (*e.g.*, diabetic air-hunger and Cheyne-Stokes breathing) and hysterical dyspnœas (tachypnœa), some of these categories indicating a morbid anatomical, some a morbid physiological change and some both. Though the two common factors, *viz.*, oxygen-lack and motor disturbance play some part in the most of these one should also find in respect of each of them definite peculiarities of breathing of very constant kind and clinical value. He dealt with the clinical features of some of the varieties of dyspnœa mentioned above. By taking of the history of the case and clinically examining the patient along the lines suggested above the diagnosis becomes often easy. The concluding opinion should, whenever possible, require symptoms to fit history and signs to conform with both. He concludes by observing "Eyes without the microscope, ears without the stethoscope, wits without the help of chemistry or radiology—not that we should deny ourselves the proper use of these—can often carry us a long way."

Prognostic Significance of the Blood Urea Nitrogen following Haematemesis or Melena

Schiff and others (*Am. J. Digest. Dis.*, 1942:9:110) give in the following lines the summary of their observations:

A blood urea nitrogen concentration of less than 30 mg. per cent in patients with hæmatemesis due to peptic ulcer, hepatic cirrhosis or undetermined cause is a favourable prognostic sign.

The presence of a blood urea nitrogen content of 50 mg. per cent or more following hæmatemesis or melena is accompanied by a fatal outcome in one-third of cases. An elevation of 70 per cent or more is accompanied by a fatal outcome in two-thirds of cases.

Elevation of blood urea nitrogen is particularly ominous following hæmatemesis in patients with hepatic cirrhosis.

The presence of shock increases the mortality rate in patients with azotæmia following hæmatemesis or melena.

In the presence of a single hæmorrhage a blood urea nitrogen curve which reaches its maximum within normal on the third or fourth day is almost always a favourable prognostic sign.

In the presence of repeated hæmorrhage a blood urea nitrogen curve which continues to rise or remains abnormally elevated is usually associated with a fatal outcome.

Angina Pectoris Treated with Testosterone

Lesser (*New England J. Med.*, 1942:51) reports on the treatment of angina pectoris with testosterone in 20 men and 4 women of ages varying between 40 and 77 who are having at least one attack of angina daily, majority having several attacks. To them every 2 to 5 days the author gave an intramuscular injection of 25 mg. testosterone proprionate in 1 c.c. sesame oil—the number of injections varied but averaged about a dozen. No untoward reactions were noted. Favourable results were obtained in all cases. The frequency, severity and duration of the attacks were diminished and the patients were able to increase their physical activities to a considerable degree without causing pain. The beneficial effects persists for 2 to 12 months after the injections. Six consecutive injections of plain sesame oil to 5 patients before the testosterone treatment was started failed to show any improvement. Two cases first treated with testosterone with improvement returned with symptoms after 4 and 7 months respectively and were given sterile sesame oil without effect, but when testosterone was given relief was obtained. A lowering of the blood pressure was noted in majority of the patients during the course of treatment; fluoroscopic examination, kymograms and electrocardiograms did not show any uniform changes due to the therapy.

Histamine for Treatment and Prevention of Serum Sickness

Cherry and Prickman (*Proc. Staff Meet. Mayo Clin.*, Ref. *Gen. Practitioner*, 1942:12:331) write that until recently no single agent has proved very efficient in either the prevention or the treatment of this condition, although epinephrine, ephedrine and certain sedatives have been of some advantage in controlling symptoms after serum sickness has developed. For the past year, we have given histaminase orally to patients who received serum therapy. Our clinical observations indicate that histaminase is of both therapeutic and prophylactic value in cases of serum sickness. Of the 21 patients on our service who have been treated with this tissue extract, 112 received therapeutic doses after serum sickness had occurred. Of the 12 patients, 10 showed definite improvement after the administration of histaminase, some of them exhibiting a remarkable response. Two apparently obtained no relief. In the majority of cases, treatment was started within 24 hours after symptoms of a reaction had become apparent. At first, the usual prescribed dose of histaminase varied from only 3 to 6 tablets or capsules daily, each tablet or capsule representing 5 detoxicating units of histamine hydrochloride. This treatment was continued an average of 4 days, but in some cases the drug was administered for much longer periods without ill effects. There were 9 patients to whom histaminase was given orally in an effort to prevent the occurrence of delayed serum sickness. In all but 2 of these cases there was either a strongly positive cutaneous reaction to the intradermal test or some personal or family history of allergy. Histaminase was given to 2 patients who had repeated administration of serum. In 4 of these 9 cases, no serum reaction developed while in the 5 others only mild transitory reactions occurred which usually disappeared within 24 hours. It is now felt that larger doses would have eliminated these reactions entirely. Two patients presented very good controls, since both of them had suffered quite severe reactions from previous injections of antitetanus serum.

SURGERY

Carcinoma of the Pancreas

Berk (*Arch. Int. Med.* Ref. Editorial, *Am. J. Digest. Dis.*, 9:113) writes that the cardinal symptom of the diseases is not painless jaundice as is taught by most of the text-book writers. His observations show that only one out of four patients presented the symptom on admission. Pain is the commonest complaint. As a primary complaint it was found in 65 per cent of the cases; it was present at some time in 82 per cent of cases, whereas jaundice appeared only in 71 per cent; when jaundice and pain were both present, the pain preceded the jaundice in about 85 per cent of the cases. The pain was variable in nature—it was sometime continuous and dull, at other times colicky, and severe, frequently radiating into the back and some times limited to the back; it was usually worse at night when the patient was lying down, being sometimes relieved when he sat up or walked about; it sometimes had an ulcer-like rhythm throughout the day. The disease progress fairly rapidly. Loss of weight is usually rapid and severe. Weakness was present in three out of four cases and nausea, vomiting and loss of appetite were found in from 40 to 50 per cent. Constipation was found more often than diarrhœa. The gall-bladder could be felt in 42 per cent of the jaundiced patients and in 24 per cent of all cases of carcinoma of the pancreas studied. Usually the liver was palpable but a pancreatic mass could be felt in only one in eight cases. Ascites was found in only about one in nine. An impaired sugar tolerance curve was found in 78 per cent and glycosuria in 24 per cent. Anæmia was usually mild. Occult blood was found in the stools in 27 per cent and fatty stools in only 4 per cent. Rœntgenologic signs of the disease were found only in about half the cases.

Factors in the Mortality of Appendicitis

Mulsow (*Am. J. Clin. Path.*, 1941:11:777) in reporting on the analysis of the factors responsible for the mortality in 122 cases of appendicitis observes that of this number, acute, gangrenous, perforating and abscessed appendicitis accounted for 97 and of this number 39 appendicitis had concretions in the lumen and 20 lay retrocecally. 17 deaths followed the removal of non-inflamed appendices. In this series of 17 cases 6 died of peritonitis, 3 of pneumonia, 2 each of paralytic ileus and embolism and 1 each from anæsthesia, from septicæmia, coronary occlusion and surgical shock. Laxatives, Christian science and chiropractic played a certain role in the total series. Delay by the patient and the family was likewise met with. But the presence of concretions in three-fourths of the perforated appendices and in one-half of the gangrenous ones, seemingly was the most important factor of all.

Serious Injury to the Rectum from Improperly Administered Enemas

Ballon and Goldbloom (*Canad. M.A. J.*, 1941:45:345) write that the possibility that the rectum may sustain serious

injury as the result of improperly administered enemas is not well enough recognised. The most essential factor in the production of such injuries is the use of a hard nozzle several inches in length. Since such nozzles are attached to syringes or bulbs, a second factor of importance is the pressure with which the enema is given. Sucking the rectal mucosa into the opening of the tube causes the injury. Local factors such as hæmorrhoids may contribute to the production of this accident. The experience of the person giving the enema also may be a factor. Following the injury the injected fluid may escape into the peritoneal cavity or the fluid under pressure may dissect the anal and rectal mucosa over a large portion of the bowel. The injected fluid may follow the fascial planes and sloughing of the rectal mucosa may occur. Enema fluids, too hot or containing soap too strong with alkali, have also been known to produce extensive injury. The tube should not be too stiff or have a terminal opening with too sharp a terminal edge. Several cases from the literature are cited and one case of the author is reported in detail.

Specific Treatment (Type A Antiserum) of Staphylococcal Septicaemia

Julianelle (*Ann. Int. Med.*, 1942:16:303) gives in the following lines the summary of his observations on the treatment of 98 cases of staphylococcal septicæmia and 4 of severe infection without septicæmia, treated with specific type A antibacterial serum prepared in rabbits (in addition to antiserum, treatment included surgical drainage and supportive care):—

Of the patients treated 48 survived and 54 died; in the latter group there were 5 patients who, although dying of other causes, recovered from the staphylococcal septicæmia.

Type A antiserum exerted its greatest effect in osteomyelitis present with septicæmia; 26 patients were treated and 23 recovered and 3 died; an additional patient with bacteriæmia recovered.

The least promising results were observed in cardiac infection and sinus thrombosis; there were 18 patients in the former group and 3 in the latter, all of whom died.

Of the 54 patients dying, 15 succumbed within 72 hours after starting serum; this and other conditions suggest that failures may not have been entirely due to impotency on the part of the antiserum.

The data indicate at this time a twofold increase in the usual survival rate, but the actual evaluation of the serum will have to await further work from different sources.

Spontaneous Fracture following Roentgen-Ray Treatment

Hight (*J. Bone & Jt. Surg.*, 1941:23:676) reports of a case of fracture of the femoral neck, being the first case in a series of 1084 cases receiving roentgen-ray treatment over the pelvis (an incidence of 0·09 per cent). The fracture resulted from weight-bearing in the presence of marked osteoporosis, developing in a bone whose blood supply had first been impaired by arteriosclerosis, and was then further damaged by roentgen-ray therapy and fibrosis. There has been no callus formation demonstrable by roentgenograms though it is three years since the occurrence of the fracture. The author holds that when roentgen-ray treatment is given over the pelvis, the portals should be placed so that they do not include the region of the femoral necks; if this is unavoidable, the portals should be as small as possible.

Sulphanilamide in the Treatment of Erysipelas

Siegel and others (*Ann. Int. Med.*, 1942:16:262) review the literature on the subjects and in reporting on a series of 303 cases of erysipelas treated with sulphanilamide at the Los Angeles County Hospital observe that the average blood level in 121 cases was 5·8 mg. per 100 c.c., that the average period of hospitalization was 8 days, the average time for the lesion to regress was 50 hours for all patients treated and this approximately the average for all age groups. The gross mortality was 1·3 per cent for the series, which is the lowest mortality rate reported in any series to date of writing. The sulphanilamide drugs are the treatment of choice in erysipelas.

Triple Dye Treatment of Burns

Dreosti (*South African M. J.*, 1942:16:181) in reporting on the treatment of 59 cases of severe and extensive burns in the Far East Rand Hospital with the triple dye method observes that only two patients died (one died on the night of admission of right apical pneumonia and the second died on the 3rd day of burn toxæmia as was found by autopsy in both cases), that the method is simple and safe requiring no use of general anæsthetic, that the mortality rate is no higher than with tannic acid or other forms of treatment and that it can be safely used on the hands and fingers in view of the thin pliable tan formed. The method used was as follows:

1. Sedative on admission—morphia, omnopon, nepenthe, etc. 2. With the patient under the influence of the sedative the burnt and adjoining areas were cleaned. This was achieved by washing with warm spirit soap followed by saline. Blisters were opened and loose skin snipped away. At no stage rubbing of the burnt area with brushes, gauze, etc. permitted, nor was ether, spirit, etc. used. 3. The local application was triple dye. This consists of a solution of gentian violet 2 per cent, brilliant green 1 per cent, made up in watery solution of acriflavin 1 in 1000. The dye was painted on with cotton wool and allowed to dry. As a rule a second application was made in 12 to 24 hours. 4. The burnt area was protected from pressure and warmth provided by cradles fitted with electric light bulbs. 5. Slough: as pointed out, this problem was dealt with as it arose, on general surgical lines. For second and third degree burns the method proved excellent. In more severe burns the same problems of sloughing tissue arose as in other forms of treatment.

First-Aid Treatment of Phosphorus Burns

Godding and Notton (*Brit. M. J.*, 1942:1:433) give below a first-aid treatment of phosphorus burns which has been found to be efficacious:

The bulk of free phosphorus and superficial acid should be removed with an alkaline powder consisting of 10 per cent heavy magnesium oxide, 5 per cent borax and 85 per cent sodium

bicarbonate. If water has been used to extinguish the phosphorus, the powder is sprinkled on wet surface and more water is added if necessary to work up to medium thick paste. The whole area of the burn should be covered and the surrounding area, if suspected of contamination. The paste is washed or wiped off and then reapplied so long as effervescence persists. *Liquid suspected of containing phosphorus must never be dabbed with handkerchief or dry material.* The whole of the remaining free phosphorus is then immobilized by the application of a glycerine-copper sulphate paste, which is applied sparingly but rubbed in well, and can be removed by washing or wiping as soon as all odour of phosphorus has disappeared from the wound. The formulæ for the pastes, either of which may be used, are:—

Copper sulphate	22·5 per cent.
Glycerin	67·5 „
Starch	5·0 „
Water	5·0 „
or	
Copper sulphate ..	20·0 per cent.
Glycerine	40·0 „
Starch	7·5 „
Sodium tauroglycocholate ..	1·0 „
Water	31·5 „

OBSTETRICS AND GYNAECOLOGY

Procaine Hydrochloride Infiltration in Obstetrics

Bickers (*Southern M. J.*, 1942:35:17) in reporting on the use of procaine hydrochloride infiltration in obstetrics observes that 1 per cent solution is used for combination of nerve block and tissue infiltration and that in the series under review there were maternal deaths and the postpartum morbidity was the same as that reported with other types of anæsthesia. Delivery was spontaneous in 79 cases and low forceps were used in 103 cases, high forceps in 22 cases, episiotomy in 133 cases, breech extraction with Keilland forceps to the aftercoming head in 3 cases and the anæsthesia was entirely satisfactory for all these procedures and the only case in which it was insufficient was an internal podalic version. 3 infants were stillborn, 2 in cases of severe pre-eclampsia and 1 a monster. There was no need of removing the placenta manually and there was no case of excessive hæmorrhage. The author maintains that with this method of anæsthesia the fœtal and maternal risks are reduced to a minimum.

Sulphanilamide Impregnated Uterine Packs

Anderson and others (*Am. J. Obst. & Gynec.*, 1942:42: 410) in reporting on the observations on the effect of packing the puerperal uterus with gauge soaked in sulphanilamide solution observe that bacteriologically more blood agar plates with no growth were obtained from the sulphanilamide packs, that there was a marked diminution in the growth of hæmolytic streptococcus ærobically and no growth was obtained in the anærobic culture, that both hæmolytic and non-hæmolytic B. coli and the spore formers were absent ærobically and anærobically in the sulphanilamide packs, and that there was complete lack of offensive odour both about the patient while the pack is in the uterus and also on withdrawal. The method of preparation of the pack is as follows: A 36-inch wide plain gauze is folded into 16 complete folds, thus providing for strip of 2½ inches wide for insertion into the uterus—the selvedge edge is on the inside. The roll of gauze is soaked in water and excess wrung out to the point when no water drips. The pack is then opened, placed upon a flat surface and the contents of two 5 gm. ampoules of sulphanilamide are distributed equally and thoroughly. The sulphanilamide is then rubbed into the meshes of the gauze to ensure even distribution throughout the length and width of the pack. The pack is then rolled upon itself and sterilized by autoclave at 225°F for 30 minutes under 20 pounds pressure.

Radium Treatment of Uterine Bleeding Caused by Benign Lesions

Randall and others (*Am. J. Obst. & Gynec.*, 1942:43: 377) report on the use of radium and rœntgen rays in the treatment of uterine bleeding due to benign lesion in 196 women. The rœntgen rays were employed in 21 cases of functional bleeding. In the entire series the bleeding was satisfactorily controlled by the initial treatment in 134 cases (68·4 per cent); in 13 cases (6·6 per cent) additional radiation therapy was subsequently required. This gave good result in 147 cases (75 per cent). In 34 cases (17·7 per cent) hysterectomy was necessary. 18 patients (9·1 per cent) complained of menopausal symptoms resulting from radiation therapy. There were 6 pregnancies in 106 patients who were married; there were 2 miscarriages, 2 premature labour with death of the infant, 1 normal delivery and 1 not delivered at the time of the follow-up. No information was available about the fœtal abnormalities. Of the 125 patients treated with radium under 40 years of age, 21 (16·8 per cent) established a 'fairly normal' menstrual cycle after radiation. One patient died from pulmonary embolism after dilatation, curettage and intrauterine application of radium. The percentage of good results with radium treatment increased with the age of the patients; the dosage of radium employed also tended to increase with the age of the patient. Intrauterine radium or rœntgen-ray therapy is the method of choice in women over 40 with uterine bleeding not associated with fibroids or with fibroids if the uterus is not enlarged to more than the size of a three months' pregnancy. In younger women radium may be a conservative form of treatment if bleeding can be controlled with 250 mg. hours or less but hysterectomy is preferable if it permits the preservation of the ovarian function and at least delays the onset of symptoms of menopause.

PEDIATRICS

Combined Immunization of Infants against Diphtheria, Tetanus and Whooping Cough

Lapin (*Am. J. Dis. Child.*, 1942:63:225) gives in the following lines the summary of his observations on the immunity response of 78 infants from 6 to 9 months of age to a combined immunization with clear diphtheria toxoid, alum-precipitated tetanus toxoid, whooping cough vaccine and whooping cough toxin in five monthly injections.

1. Diphtheria immunization resulted in negative Shick reactions for 100 per cent of the subjects, 97 per cent having above 1/250 unit of antitoxin per cubic centimetre of blood and 91 per cent having above 1/10 unit per cubic centimetre.

2. Tetanus immunization resulted in an increase of the average antitoxic level to 0·50 unit, with 93·2 per cent of the subjects having 3 months after immunization an antitoxic level greater than is attained by the usual prophylactic injection of tetanus antitoxin. Evidence has been cited to show that 6·8 per cent who had titre below this level at the three month period following immunization have a sound basal immunity and on exposure will respond to a boosting dose with a safe antitoxic level.

3. Whooping cough immunization resulted in positive reactions to agglutination tests for 100 per cent of the subjects, complement fixation of 4 plus for 87·2 per cent and positive for 97·4 per cent, and a positive mouse protection titre for 87·5 per cent.

Bronchoscopic Aspects of Asthma in Children

Van Loon and Diamond (*Am. J. Dis. Child.*, 1942:63:217) in reporting on a bronchoscopic study of 100 children with chronic allergic asthma observe that there was no evidence of bronchial spasm or œdema in any of the patients, that there were present in 91 cases nonspecific inflammatory changes, in 36 cases accentuation of the physiologic expiratory narrowing and in 60 mucopurulent or thick mucoid secretion and that there was no characteristic bacteriologic picture and no lobar or segmental atelectasis in any children. Of 20 patients who were bronchiographed 5 showed bronchiectasis. The authors further observe that the only constant feature of endoscopic appearance in children examined in the acute stage was the presence of thick viscid intraluminal secretion.

Maningitis due to Escherichia Coli

Barret and others (*Am. J. Dis. Child*, 1942:63:41) in reporting 2 cases of meningitis due to *Escherichia coli* with recovery following the treatment with sulphanilamide and derivatives and in reviewing the literature on the subject observe that meningitis due to *Escherichia coli* is most common in infants under 3 months of age, that without chemotherapy the fatality rate is about 80 per cent, that the portal of entry may be obscure but associated infections of the middle ear, gastrointestinal tract, urinary tract and umbilicus are recorded, that bacteriæmia has been present in a number of cases in which blood cultures have been made, that the course tends to be protracted with relapses and the development of hydrocephalus, that the spinal fluid continues to be infected for several weeks, that sulphathiazole and repeated number punctures are the methods of choice in treatment (since *Escherichia coli* is one of the few oragnisms which can grow freely in cerebrospinal fluid and sulfathiazole is more effective than the other drugs of the group in inhibiting the growth of the organism in C.S. fluid) and that it is possible that the whole blood transfusions in infants under 2 years of age may aid in the treatment. The authors maintain that if an adequate concentration of sulfathiazole in the cerebrospinal fluid cannot be obtained, sulphadiazine should be administered.

Treatment of Bronchial Asthma in Children

Brown (*New England J. M.*, 223:629, Ref. *Am. J. Dis. Child.*, 1942:63:420) writes that acute attacks of bronchial asthma in children can be treated by any of the sympatheticomimetic drugs of the ephedrine group. Ephedrine sulphate is the best; the usual dose is ⅜ grain. However, it has undesirable side actions and it is necessary that additional sedatives be given. The best of these is phenobarbital since many of the barbiturates are vasospasmodic. It may be given in doses of ¼ or ½ grain. For acute attacks of greater severity, small doses of epinephrine hydrochloride solution (1 in 1000), 2 or 3 minims may be injected subcutaneously. The dose may be repeated over 15 minutes for 4 doses, if necessary. In cases in which ephedrine sulphate given by mouth is not necessarily effective by itself it will enhance and prolong the action of the epinephrine. Rectal medication seems effective in children who cannot take the drug orally. In the severer attacks, epinephrine base in oil, 0·5 to 0·8 c.c. of the usual preparation (2 mgm. per c.c.) should be given every 12 hours. Status asthmaticus is not common in children, but when it does occur it requires the treatment given to adults. Theophylline with ethylenediamine administered intravenously is used, the dose being 3½ grains; 50 c.c. of a 50 per cent solution of sucrose is given at the same time. Epinephrine hydrochloride solution, 1 in 1000, may also be given intravenously with this injection, this dose being 0·1 or 0·2 c.c. Ether in olive oil (30 c.c. of ether to 60 c.c. of olive oil) given rectally will also help to break a severe asthmatic attack. Morphine in any dose and in any form had a deadly effect on an asthmatic child; it should never be used under any circumstances whatsoever. Epinephrine given by inhalation has been utilized, but it is very difficult to teach a child inhalation therapy. For children with the extrinsic type of asthma, a change in environment is imperative. For those with the intrinsic type, any intercurrent infection can be cared for more easily in the hospital than at home. For those with continuous chronic asthma any of the several remedies containing potassium iodide and tincture of stramonium may be employed. The general care of the child still remains the most vital factor.

A PRELIMINARY REPORT ON THE TREATMENT OF ANGIOSPASTIC CONDITIONS WITH ERGOT AND NICOTINE

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(Continued from Vol. XI: No. 12: page 391)

Phase Actions are represented by Wilder's [136] and Danielopolu's [137] extensive works on the autonomic nervous system. The latter's works in short are that a predominant action on the sympathetic or parasympathetic depends upon the intensity of action of the exciting factor. A small dose of adrenalin is amphotropic with predominance on the parasympathetic, a large dose is amphotropic with predominance on the sympathetic. The action of ergot in small doses is similar to adrenalin (in small physiological doses) as reported by Samson Wright [138], and the high dilution subphysiological doses in this sense has not, with regard to ergot, been experimented with, to my knowledge. A weak excitation of the carotid sinus produces a reflex which predominates over one group, while strong excitation produces a reflex predominating over the other. Although these results may be obtained in the healthy, they are even more easily studied in the presence of pathological conditions. Another example of phase effect is seen in the following: Alypin applied to normal blood vessels (1:4000) causes vasoconstriction. But should the same drug be applied first in a strength of 1:2000 there is constriction, then a dilution of 1:4000 applied subsequently will create vasodilatation. Similarly, Zih reported at the Physiological Congress, Frankfurt, 1927, that spleen extracts in small doses produce an increase of erythrocytes, while large doses decrease, and Sollmann, in his well-known text book of pharmacology, mentions in passing at least 125 well-marked instances of reversible effects.

Finally, since, to my knowledge no work on ergot has been done on these lines, we shall have to limit ourselves to extensive work on phase reactions of adrenalin. According to Cushny [139] "ergot action resembles that of adrenalin and it also acts on the myoneural junction of the sympathetic nerve." Adrenalin has definitely biphasic action: early increase of blood-calcium and sugar, decrease in blood potassium and phosphates, later the reverse [Vollmer (140 and 141)], early hexo-phosphoric acid decrease and then increase [Lawacezak (142)], increase of blood sugar followed by decrease [Brosamelen (143) and Gyorgy and Herzberg (144)], in tetanus and in fever hyperglycæmia decreased and then predominated [Pollack (145)], there are also additional blood phases [Ederer (146)], dilution of blood followed by over-compensation, hypochloræmic phase followed by hyperchloræmic state, retardation and then augmentation of gastric movements [Danielopolu (147)], in sympathicotonics it retards the gastric secretion, in vagotonics it augments [Mahler (148)]; stimulation and depression of the vasomotor centre have been reported [Pilcher and Sollmann (149), Brown (150)], variations of dilator response of blood vessels is also known with dilute solutions, prolonged perfusion and change of tonus [Brodie and Dixon (151), Sollmann (152), Ogawal (153)]. Skeletal muscle arteries are dilated by small doses and constricted by large [Harman (154)], coronary constriction from small doses which do not affect the heart muscle [Meyer (155)]. Very dilute solutions depress the heart, large amounts stimulate, still larger depress; stimulant concentration remove the Luciani irregularity, lower concentrations increase it [Sasaki (156)]. In pregnant cats adrenalin produces contractions of the uterus, in non-pregnant ones relaxation [Dale (157)].

While Spiro [158] denies that ergotamine tartarate can produce gangrene because it is not a vasoconstrictor, and Straub [159] says that the gangrene is due to vasodilatation, Jacobi [160] has shown the prolonged vasoconstrictor action of whole ergot. v. Storch [161] blows hot and cold in one breath, maintains that E. A. Carmichael has definitely shown the vasoconstrictor action of ergotamine in sympathectomised blood vessels of the finger, and further quotes Pollack's experiments in ergotism that gangrene is hastened by section of the abdominal sympathetic chain! He admits that "experiments in animals and man and pathological evidence thus confirm the impression that at least gangrenous ergotism due to ergotamine tartarate is a vasospastic obliterative process"

.....furthermore, ergotamine tartarate is capable of producing either type of ergotism in animals and man. It would therefore appear that ergotamine tartarate produces a type of ergotism as yet indistinguishable from whole ergot" (p. 298).

It would not be wrong to assume, that should experiments be carried out with ergot on the basis of high dilutions, we shall get in accordance with Wilder's law of "Original value response" and Danielopolu's observations and Kotschau's rule of typical effects, the opposite of Cushny's observation, *viz.*, "ergot stimulates the myonetural junction in small doses and paralyses it in large" (p. 545). Actually Cushny's small dose is the medium dose of Wilder, Kotschau and Danielopolu, and corresponds to Kotschau's second phase. In experimental work with ergot, it is not improbable that the weakest solutions (high dilutions) will give vasodilatation, the medium strengths vasoconstriction, and the strong doses vasomotor paralysis. Some pharmacologist should investigate that aspect.

In my earlier clinical experiments, I was not led to try my experiments on the basis of these arguments, but solely to test the homœopathic thesis that likes cure likes. Perhaps the best example is Wapler's [162] proof that cats pretreated with small doses of strychnine are immunised against tetanus toxin. The pharmacological action of strychnine and tetanus toxin are identical in their point of attack [Samson Wright (163)], and I know definitely of one case (the son of a doctor) where antitetanic serum had failed and the child was saved by repeated doses of homœopathic strychnine (1:1,000,000). It is futile to consider hypotheses, however ingeneous, as equivalent to explanations. The only feasible hypothesis, which still does not amount to an explanation, is that in the body, the pathological break-down of amino bodies may create a toxic condition similar to ergot. It is futile to search for quantitative amounts in the body. Actually, we shall have to consider the affected human subject as reacting with allergic manifestations to toxins. In other words he is highly toxin-sensitive to the smallest amount of toxin to which the normal individual is insensitive. Zimmer

[164], who was Bier's assistant has shown that a gouty patient is 250,000 times more sensitive to an unit dose of formic acid than a non-gouty subject. This proves also Schulz's original thesis that the pathological tissue is sensitive to very much smaller stimuli than the normal tissue where he states:

"the activity of a drug depends upon the existence of a connection between it and the tissues upon which it acts; the action is in accord with Pfluger's law; under pathological conditions the sensitivity is increased so that what is weak" and what is "strong" depend upon the individual case." [Schulz (165)].

If this view is accepted that the angiospastic conditions are a form of allergic manifestation against complex toxins, it will be evident that merely administration of vasodilating drugs must fail, as they have, in experience. The problem is to find an allergen identical with or proximating to the totality of the allergic manifestation and thus to desensitise the individual manifesting them. Sufficient evidence has been brought forward to show the extraordinary similarity between the reactions of the human organism to chronic ergotism and the allergic manifestations of the human body to unknown toxins. It stands to reason that in the absence of our knowledge of the exact and specific toxins, desensitisation shall have to take place with a known antigen creating the same symptoms. Storm van Lecuwen [166], an international authority on the subject of allergy, states."

"all methods of specific treatment of the allergic diseases are based upon the same principles, namely that injections of very small amounts of allergens can prevent the breaking out of an attack that has been caused by this allergen."

Whatever highly scientific and fancy name we may give it, the principle approaches very suspiciously close to homœopathy. For instance, Bier [167] has treated x-ray burns with small doses of x-ray, so also he introduced the treatment of post-operative ether bronchitis with intramuscular injections of small doses of ether in olive oil [168], and Schneck extending Bier's principle, the conscious application of the similie principle, reports a series of 300 cases of the crushing of the upper part of the body, in which not a single case was lost through secondary pulmonary complications [169]. Applying the same principle, Richter [170] treated neo-salvarsan dermatitis and other manifestations of arsenic poisoning with small doses of the same drug in six cases with complete success, and I myself have rapidly relieved a case of accidental paravenous Mapharside inflammation with arsenic thirty centismal dilution.

Thus ergot must, at present, be considered the nearest approach to the allergen. This is all the more understandable when we examine the amazing complexity of the constituents of total ergot, not a single one of which has the rapidity of action of the total ergot (Sollmann), and that most of them have equivalents in the amines disintegrated in the human body through intestinal bacterial activity [Abderhalden (171), Guggenheim (172), Feldberger (173)]. The most important are ergotoxine (Barger), ergotamine (Stoll and Spiro), cadaverine, isoamylamine, tyramine, histamine the most important among the amines, choline and acetyl choline, leucine. Acetyl choline has a muscarine-like action. Handovsky [174] showed the biphasic action of histamine, small doses stimulating and large doses paralysing and killing the organism.

In nicotine (tobacco) isoamylamine from leucine is found as in ergot, and its action on the vasoconstrictors is similar and therefore, gains considerable importance. Nicotine does not lead to gangrene but to arteriosclerosis and vasospasms. It affects the function more than the structure. Unfortunately, standard books on pharmacology make practically no mention of studies on the action of tobacco. Recent literature on the possible relation and influence of tobacco or nicotinism to thromboangitis obliterans has shown the considerable importance of nicotine as a vosoconstrictor [Lampson (175), Maddock (176), Barker (177), Wright and Moffat (178), Johnson (179), Herrel and Cusick (180)]. The action of nicotine on the coronary arteries and the peripheral vessels was known to the homœopaths long ago especially through the extensive work of Cartier in France. The symptomatology of nicotine is exceedingly well marked: nausea, vertigo, death-like pallor, icy coldness and sweat, intermittent pulse, complete prostration of the entire muscular system, collapse, gastralgia, enteralgia. Produces high tension and arteriosclerosis of the coronary arteries. It is one of the best homœopathic drugs for angina pectoris, with high tension, hard cordlike pulse; sick headaches, central scotoma, amaurosis, muscæ volitantes, rapid blindness without lesion followed by venous hyperæmia and atrophy of the optic nerve; tachycardia, bradycardia, acute dilatation of the heart after exercise; hands and legs very cold, trembling, paralysis, tabetic gait, alternate tonic and clonic spasms, head drawn back, contraction of eyelids and masseter muscles, rigidity of muscles of neck and back, spasms of the laryngeal and bronchial muscles [Boericke (181)].

The biphasic action of nicotine is well-marked: It first stimulates and then paralyses the ganglia on the vagus, the ganglia on the vasoconstrictor nerves, the ganglia on the chorda tympani and all the sympathetic ganglia. It first stimulates and then paralyses the respiratory centre, it alternately causes a tetanic contraction of the muscular walls of the stomach and of the entire alimentary tract and then relaxation. It similarly tetanises the bladder. The pupils are first contracted and then dilated. It is a functional rather than an organic poison. [Stoneham and Neatby (182)]. Spasms and convulsions, constant hunger, cardialgias, spasms of the limbs, numbness and coldness, alternating with burning in the arms and legs (Hull's Jahr, Symptomatology). Thus the actions of ergot and nicotine are similar in functional disturbances. The action of acetyl choline resembles that of small doses of nicotine [Samson Wright (183)].

Report of Clinical Cases

I am indebted to Dr. E. B. Christian of the Zaheerabad Leprosy Investigation and Treatment Centre for conducting the experiments on 4 cases (Cases 1-4) of circulatory disturbances in leprous patients. The remaining cases are non-leprous.

Case 1. N. D., 25 years Hindu, history of leprosy 4 years, classification LI, NI. Since one year intolerable burning, tingling, stinging and formication in hands and feet. Hands and feet bluish red, towards evening swelling of fingers and toes with increase of pain. Kahn test 1+. No history of V.D. Sedimentation index 20 mm. Previous treatment: neosalvarsan 6 injections, gelsemium, bromides, pot. antimony tart, 12 injections—temporary relief. On my recommendation he was given

ergot 6th centismal dilution 3 doses. Complete relief. Relapse after 3 months. This time ergot 30th centismal dilution was given, 3 doses at 2 hour intervals. Complete relief, disappearance of swelling and discoloration. Duration of observation 1 year.

Case 2—A. L., 28 years, Mohamedan, leprosy 8 years, classification N1, N2. Burning sensation of hands and feet, better immersing in cold water, more in the day when the sun is hot, numbness, tingling, formication and sensation of heaviness in the fingers. Kahn test negative. Sedimentation index 10 mm. Ephedrine and Gelsemium failed. Ergot 6th centismal dilution 3 doses. Complete relief. Period of observation 1 year.

Case 3—Y. E., 40 years, Hindu, leprosy 16 years, classification N2 Kahn 3+. Sedimentation index 30 mm., Trophic ulceration right foot. Numbness, formication, shooting pains, spasms in the calves. Avenyl injections for 1 year. Kahn 2+. Gelsemium and bromides failed. Ergot 6th centismal dilution 3 doses, complete relief. Duration of observation 1 year.

Case 4.—M. S., 40 years, Hindu, female, leprosy 5 years, classification LI. Khan test negative. Sedimentation index 25 mm. Burning, stinging pains in hands and feet, livid discoloration acral parts, pains intolerable at night. Gelsemium, bromides and ephedrine without success. Ergot 6th centismal 3 doses with complete relief. Duration of observation 13 months.

These paræsthesias in leprous patients is a very distressing symptom and so far in official leprosy treatment we have been powerless to ameliorate them. In ergot we are thankful to have found an effective and cheap remedy for this distressing complaint.

Case 5—S., 27 years, cinema operator. For three years had developed ulcerations at the tips of fingers and toes which were treated at outpatient hospitals with ointments. No relief; was given 6 injections of collosal manganese and 6 of colloidal sulphur, and a colleague took infinite pains to dress the ulcers daily. The associated findings were: lividity of the toes and fingers, extreme coldness to touch, brittle nails, numbness, tingling, unbearable spasmodic pains, better washing hands in cold water. Ergot 30th centismal dilution 3 doses brought him complete relief. Slight recurrence of pains after 4 months were put right with 3 doses of ergot 200th centismal dilution, all sensations normal. Duration of observation 2 years and 4 months.

Case 6—R. R., 23 years, came to me from the districts with ulceration of the distal digit of the middle finger of the right hand. Had been incised by the district civil surgeon for alleged whitlow. The affected digit was sloughing, blackish, with fetid greenish pus, the lividity spreading to the base of the phalanx. The index and third fingers were also cold to the touch, bluish, œdematous and painful. The index and middle fingers also of the other hand were painful and bluish, and the patient felt pain, numbness, swelling, cramps and spasms in them. He was given ergot 6th centismal one dram of pills, 2 pills three times per day till improvement should begin. From the second day all spasms and pains disappeared, the ulceration healed with granulation and scarification and the pains and lividity in the left hand fingers also disappeared. Complete recovery with atrophic scarification of the ulcerated finger.

Case 7.—Veerappa from the districts, 45 years. Gangrene of the right foot. Great toe sloughing with line of demarcation covering the lower half of the dorsum of the foot. Discoloration, bluish, livid, black in spots, great toe looks like a charred stump with thick greenish pus oozing out of it. Agonising pains kept the patient crying day and night. He was bathing his foot with iced water when I saw him for the first time. Dorsopedal pulsation absent, popliteal pulse not determinable. Clinical diagnosis: Thromboangiitis obliterans. Duration 4-6 months (exact time not elicitable, since the patient was in a maniacal condition owing to pains). Previously pains in the lower leg from knee down to sole of the foot, increasing and subsiding after long intervals. 3 doses of ergot 6th centismal dilution were dissolved in half a cup of boiled and cooled water one teaspoonful as dose every hour relieved the pains within 12 hours. The medicine was continued at 6 hour intervals the next day and only twice a day for the next 4 days. The discoloration slowly improved with total absence of pain. Drops of blood appeared at the end of the sloughing skin and the œdema slowly disappeared. In 3 weeks healthy granulation appeared with total disappearance of pus. After 4 weeks transient pains appeared and he was given 3 doses of ergot in 30th centismal dilution. The patient was under my observation for one year during which there was no relapse. Since then I have not heard from him.

Case 8.—K. A., 22 years, Osmania University student. Right hand, numbness, tingling, formication, sharp shooting pains, spasms, cramps, intense coldness to touch, but alternating sensations of burning and chilliness, fingers swollen and drawn apart as if ankylosed, colour bluish, cyanosed with reddishness in parts. On hanging the arm down pain is excruciating. Brachial artery pulse palpable. Condition worse since 6 weeks, previous 6 months pains came and went. Was given Padutin with temporary relief. Clinical diagnosis: Raynaud's syndrome, pregangrenous stage. Heavy smoker. Each time he resumed smoking the spasms became worse. Ergot 30th centismal 3 doses created prolonged aggravations and increased the pains. Revising the case, I took into account the aggravation due to tobacco. Nicotine 200th centismal dilution 3 doses immediately brought about relief.

According to Stoneham (p. 838), high dilutions of nicotine will counteract the evil effects of tobacco on the cardiovascular system, especially the "tobacco heart." Duration of observation 3 years. No relapse since the patient has been weaned of the tobacco habit with Caladium 30 c. and Plantago 6 c.

A parallel case has been reported by Fischer [184]. 42 year old man was treated at the Municipal Hospital, Wuppertal for Raynaud's disease. Treatment with radiant heat, diathermy, massage, suction apparatus and 200 injections of Padutin had failed. Both toes begin to gangrenate. After 3 days of raw food diet, local warmth and suction treatment, ergot 3rd centismal t.d.s was given in 10 drop doses. No result after 4 weeks of treatment. Nicotine 2 centismal 10 drops t.d.s brought about severe reaction on the 4th day. Since a reaction is a healthy sign (but not if it is prolonged—N.M.J.), nicotine 3 centismal was given 15 drops t.d.s. which was well tolerated. In 14 days the right toe healed satisfactorily, whereas the left

toe where the process had advanced too far had to be amputated. Worthy of notice was the good circulation after the administration of homœopathic remedies, which was observed during the amputation. The German School of Homœopaths operate with the lower dilutions. While I also employ the lower dilutions, I use the higher dilutions which the Germans seldom use and find they work better and deeper. I have frequently used ergot 200 centismal dilution in functional uterine hæmorrhages with outstanding success.

Case 9.—S., 28 years, electric borer machine operator. This is altogether a remarkable case which was examined at my request by Major M. G. Naidu, Dr. Bankatchandra (1st Physician, Osmania General Hospital), Dr. Sass, M.D. (Rome) and Dr. Timmaraju. This man showed a high degree of adynamia and dehydration as I have never seen before. Symmetrically on the middle and third fingers of both hands and third and fourth toes of both feet were gangrenous. A thick yellowish greenish pus oozed out of the ulcers on the fingers. The gangrenes on the toes were stone dry. There were spots of anæsthesia on the ankles and hyperæsthesia on the thigh. Dr. Christian, with wide experience of leprosy, made a careful examination and neither he nor we could find any thickened nerve, in spite of which a Leprosy Hospital made the unpardonable mistake of diagnosing it as leprosy. The unanimous diagnosis was Raynaud's disease. After one month at the Leprosy Hospital he came back worse. Ergot 200th centismal dilution 3 doses were given by me and within a very short time I was surprised to see a changed man. The gangrene disappeared with healing of the ulcers, the affected digits cicatrised with ankylosis, the adynamia and dehydration disappeared, and within a month the man went back to his job at the electric borer machine. The Lallaguda Railway Hospital has a record of this case. A year and a half later, he went to his village and I was informed by his relatives that he developed the same adynamia I had noticed and under village treatment succumbed to it.

A parallel case is reported by Gerlach [185] who treated a patient with gangrene of the toes resulting from ergotamine (Gynergen) poisoning. Zorn [186] had in a similar case to resort to Padutin and physicotherapy. Here Gerlach has proved the reversible action of ergot by treating an ergot (ergotamine-Gynergen) poisoning with ergot. Perhaps nothing proves the homœopathic thesis better. Because of the other symptoms (cramps, nausea, vomiting), and as a result of his interest in the pharmacology of histamine, this drug was given in low dilutions with the result that the spasmodic pains disappeared, and the gangrene improved. Since histamine is pharmacologically allied to ergot, he treated 6 other cases with ergot in dilutions of 1:100 to 1:1,000,000. The usual dose was 5 drops of 1:1000, three times a day followed after some days by 1:100. If aggravations took place he gave 1:1000 to 1:1,000,000. He observed 6 cases for over 5 years with very satisfactory results. Some symptomatic improvements were observed in other forms of gangrene. I myself use the higher dilution of 1:1,000,000,000,000 as the least, and have had no success in gangrenes not based on an angiospastic action, *e.g.*, traumatic gangrene. It is a homœopathic experience that when a lower dilution aggravates the higher dilution removes it. Another point to note is that the higher dilutions should be utilised for the predominantly functional disturbances and the lower dilutions for those conditions where structural changes are involved.

Case 10—52 year old Mohamedan lady. Referred by Major M. G. Naidu. Objective findings were: Pulsating aneurysm right subclavian artery, visible above the right sternoclavicular joint, enlarged thyroids, rapid pulse (140), exophthalmos, blood pressure 170-110, paræsthesia and formication down left arm, trembling and twitching of the arms on extension, great restlessness and flushing, menses stopped. Diagnosis: Postclimacteric angioneurosis and hyperthyreosis. No specific history. Great dyspnœa and inability to lie down. Ergot 200, 3 doses with vast improvement with abatement of restlessness, dyspnœa, tremors and blood pressure came down to 140-95. The flushes of heat did not decrease (climacteric disorder). This was removed by a single dose of Lachesis (Bushmaster or Surucucu snake venom) 200, with complete relief. She was observed for a period of over 2 years and after my shifting to Secunderabad I lost trace of her. I was informed a short while ago, a year after she had stopped my treatment that she died of sudden heart failure. Wapler [187] recommends homœopathic ergot 2 and 3 c., as a specific for the paræsthesias of climacteric.

Case 11.—Mrs. A., Anglo-Indian lady 72 years old, thin shrivelled, rapid pulse, irritable, volatile, changeable moods. A babool thorn pricked her thumb, festering with gangrenous outlook; was treated for two months at hospital out-door departments. On examination she spoke of formication, numbness, tingling, and burning pains. The ulcerated thumb itself was painless to a prick. Ergot 30c., 3 doses cured the thumb completely. Now at the cicatrised spot there is tenderness and pain on pressure. More remarkable is a noticeable change in her generally. She is putting on weight.

Case 12—C. E. (Indian Christian). For 4 months noticed sudden pain in calves going down to soles of the feet which forces him to stop and flex the leg or sit down. Right leg more affected. Gets attacks more on smoking. Nicotine 30, 6 doses, one dose morning and evening, has greatly improved him and for 2 years he has had no attack. He has been weaned of the tobacco habit by Caladium 30.

A parallel case has been treated by Weissenbach and Perles [188] with small doses of histamine 1:2000(0·0005) in 1 c.c. given strictly intradermal. Hypodermic injections brought about severe reactions. This almost amounts to the homœopathic principle.

Case 13—J. D., 49 years, Indian Christian, heavy smoker, repeated attacks of pain in the cardiac region radiating down the left arm. Blood pressure 158-110, changeable during attacks. Gets attacks often, nausea, breaks out in cold sweats, great weakness, anxiety and trembling during attacks. Had three attacks in one day. Attacks come off and on, especially after exertion, heavy smoking. Bowels clear, no meteorism. Diagnosis: coronary spasms, anginoid attacks. Tabacum 30c. relieved the condition completely. Has been weaned of nicotine abuse with Caladium 30.

Case 14—A Mohamedan lady, 40 years, referred by Major M. G. Naidu. Intense rightsided migraine which did not subside for over 22 days under Saridon, Luminal and Morphia.

Thin, scrawny, irritable, in great distress. Metrorrhagia, intermenstrual bleeding. Ergot 200, 3 doses, one dose 2 hourly. The relief was spectacular. This is one of my earliest test cases which converted me to faith in homœopathic therapy. No recurrence for 3 years. Since then I have lost sight of her. I have used the same therapy that is fashionable and highly scientific today, *viz.*, treatment of migraine with ergot preparations as Lennox [189] and v. Storch [190], without all the bye-effects that they have to combat against, with much more permanent results, with an infinitely smaller dose than that used by them, and on an entirely different basis—the similarity of the drug picture to the picture of the disease presented in its totality.

Conclusions

These preliminary investigations give me reason to believe that with further trials on a larger number of cases, which any hospital dealing with such cases of peripheral circulatory disturbances, should try out for further corroboration, may lead after careful sifting of the material, to the utilisation of an extremely cheap and efficient remedy. In the 14 cases that I was able to study, there was not a single failure. The relief was permanent and in all cases gratifying. Since the dosage is infinitesimal, it cannot do harm, even if it may fail in doing good. The aim of this paper is not to prove the superiority of homœopathy but merely to show the essential unity and identity of the scientific basis in allopathy and homœopathy which most members of the profession are not aware of. I have carefully avoided any reference to homœopathic literature except to draw attention to parallel findings. On the contrary this whole paper has been built upon a study of allopathic research. No definite opinion can be passed until further and larger material has been carefully studied and clinically verified. This paper merely intends to draw attention to the possibility of erogt and its allied histamine and nicotine being employed in high dilutions as being at present the nearest approach to a rational therapy on the principle of the reversibility of drug action for conditions that are similar to those created by these noxæ.

In the H. E. H. Nizam's Dominions the State recognises Ayurvedic, Allopathic, Homœopathic and Unani faculties, consequently the readers are requested to draw their own conclusions from the paper which was read at the conference at Hyderabad, Dn.—Editor.

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NOTES AND NEWS

I. M. A. MEMORANDUM ON CIVIL DEFENCE MEDICAL MEASURES FOR BENGAL

For effective and proper organisation of Civil Defence, the the first essentials are formulation of a detailed and comprehensive scheme by the Government, and willing and helpful co-operation from the public. The Government should also arrange for wide publicity of their scheme so that the people may know what the Government have arranged for, and also where and how they can co-operate for best results. Active co-operation of the medical profession will play the most important role in any scheme of Civil Defence. For these obvious reasons, the various branches of the Civil Defence Department in England have been placed under the administrative control of the Ministry of Health with the "Central Medical War Committee" of the British Medical Association acting as the official Advisory Committee on all medical matters concerning the War.

The medical organisations of Bengal, especially the Indian Medical Association have, from March 1941 onwards asked the Government to place before the medical and lay public, a scheme showing the details of medical preparedness of the province. The medical profession of Bengal has many a time voiced its unequivocal opinion that medical men are of paramount and indispensable necessity for proper management and treatment of air raid casualties, and that the medical men of Bengal are always willing, ready and eager to help the civilian air raid casualties with their ungrudging services. This opinion of the medical profession of Bengal has been voiced from different platforms, conferences and also as statements to the lay press. His Excellency the Governor of Bengal also recognised the need and importance of popular co-operation in civil defence measures and created a portfolio of Civil Defence Co-ordination with one of his popular Ministers in charge of it. The Hon'ble Minister in charge, Mr. S. K. Basu, has in turn formed a central advisory committee but very unfortunately, there is not a single medical man in this committee of 18 members (*vide Amrita Bazar Patrika* dated the 24th March, 1942). On 27-12-41 the Hon'ble Minister, Mr. S. K. Basu and the Surgeon General with the Govt. of Bengal addressed a large gathering of medical men, called at the request of the Hon'ble Minister, and appealed to them to come forward and help with their services. The Hon'ble Minister also invited the medical profession to co-operate in various spheres of medical defence activities. At that meeting, a committee was formed consisting of six representative medical men to act as a liaison body between the authorities and the medical public in general. After four months, this Liaison Committee placed the report of the work they had done before a general meeting of medical men at which the following resolution was passed:—

"The medical profession of Calcutta and suburbs at a conference held on 30-4-42 at the Calcutta Medical Club, is regretfully of the opinion, from the report of the Liaison Committee placed before the Conference, that inspite of the solemn assurance given by the Hon'ble Minister, Mr. S. K. Basu, now in charge of Civil Defence Co-ordination, Bengal, the help and co-operation of the Liaison Committee, appointed by the medical profession and accepted by the Government, has not been made full use of by the Government in dealing with the various medical aspects of civil protection of the population of Bengal in case of air raid, with the result that the organisation is still incomplete, inadequate and likely to prove ineffective when the actual emergency arises."

The Indian Medical Association has always co-operated with the Government in this and allied matters and has always insisted on a comprehensive and detailed scheme dealing with all medical aspects of Civil Defence. In March, 1942, the Bengal Provincial Council of the Indian Medical Association drafted a skeleton scheme concerning medical aspects of civil defence, and copies of this skeleton scheme were published in the press and forwarded to the various departmental heads of the Government including the Hon'ble Chief Minister, the Hon'ble Minister in charge of Civil Defence Co-ordination and also the Surgeon General with the Govt. of Bengal. In that skeleton scheme the outlines of planned evacuation, medical provisions for safer receptive areas and medical aspects of civil defence measures in the emergency areas of this province were compared to those of England. To quote one instance, while Great Britain with a population of about 45 million arranged for over 200,000 A.R.P. hospital beds at the outset of war in 1939, Bengal with about the same population had not made provision for more than 10,000 A.R.P. hospital beds by the end of 1941. In that skeleton scheme the Government of Bengal were entreated to arrange for (only) 24,000 A.R.P. beds throughout the province of Bengal. The Government were also requested to establish a number of Base, Advanced Base and Convalescent Hospitals with about 16,000 beds (out of this 24,000), and also to arrange for the dispersal of necessary medical equipments and personnel for this purpose. The Surgeon General invited the members of the Emergency Medical Services Standing Committee of the Indian Medical Association for a discussion regarding the I.M.A. skeleton scheme and also the Govt. scheme. At this meeting held on 1-4-42 the Surgeon General, on being requested, promised to send along to the E.M.S. Standing Committee a copy of the detailed Govt. scheme—which unforunately he did not do. Reminders were sent to the Surgeon General for a copy of the comprehensive and detailed Govt. scheme concerning medical aspects of civil defence for Bengal, but only "a statement showing the hospital accommodation for aid raid casualties in Calcutta and the industrial areas and the number of doctors required for the extra A.R.P. beds in hospitals" was sent by the Chief Medical Officer, A.R.P. and later we find that the Surgeon General writes to the Hony. Secretary, E.M.S. Standing Committee (No. 2188 A.R.P. dated 23-4-42) that he thinks this "ought to be sufficient for your purpose and it is not necessary for me to submit for the scrutiny of the Indian Medical Association details of the staff and equipment of the Emergency Hospitals." The above mentioned facts cleanly show that the help and co-operation sought by the authorities from the medical profession, when extended, have been treated with great indifference and discourtesy by the A.R.P. branch of the medical department.

The Indian Medical Association, therefore, realises with much regret that even at this hour of grave peril to this pro-

vince, the Government do not feel the necessity of popular co-operation in matters of civil defence. It is needless to stress again how essential this public co-operation is for the successful operation of such an organisation. However, the I.M.A. feels that, during the last eighteen months, it has done all in its power to assist the Government in formulating a comprehensive scheme. It is, now, for the Government to carefully look into the working of their present A.R.P. arrangements and to realise the importance of the "skeleton scheme on medical aspects of civil defence" prepared by the Association. At this critical stage when the enemy is knocking at our door, it is the sacred duty of the Government to bring the A.R.P. medical organisation up to the standard necessary for the protection of the people.

QUININE AND THE URGENT NEED OF RELEASE OF ITS STOCK BY THE GOVERNMENT

That there is shortage of quinine in certain places owing to freezing of the stock by the Government and that there is an urgent need of its release will be evident from the correspondence published hereunder:

Copy of letter No. SB/7-3/144 dated 30th July 1942 from the Hon. Jt. Secretary, Karachi Medical Association to the Hony. General Secretary, the Indian Medical Association, Calcutta:

I enclose herewith the copies of the correspondence passed between the President of my Association and the Director General, I.M.S. for your information and necessary action.

The question of supply of quinine is becoming very acute day by day as the Government has frozen and is likely to freeze the stock of all the available quinine from the importers. The local chemists say that they have no quinine and I have been given to understand that few sundry merchants have some quinine with them for which they quote Rs. 156/- or round about per pound to the lay public, but to the medical profession they say that they have none in stock.

This question is becoming even more acute in our Province owing to recent heavy rains and floods in Sind. The malaria incidence is increasing by leaps and bounds and to meet with this exigency quinine must be made available to the medical profession. We hope that you will kindly take up this question with the authorities concerned at your earliest.

* * * * *

Copy of the telegram addressed to the Director General, Indian Medical Services, in India, Delhi, by the President of the Karachi Medical Association (Sind Provincial Branch of the I.M.A.) Karachi:

Kindly release one hundred pounds quinine sulphate, Howard for medical profession, Karachi from stocks detained under your orders with Anglo-Thai Corporation, practitioners facing difficulties.

* * * * *

Copy of the letter No. MC/13-2/35 dated the 21st July, 1942, from the President, Karachi Medical Association (Sind Provincial Branch of the I.M.A.) to the Director General, I.M.S., Delhi:

In continuation of my telegram dated yesterday, "Kindly release one hundred pounds of quinine sulphate Howard for medical profession, Karachi, from stock detained under your orders with Anglo-Thai Corporation, practitioners facing difficulty," I have the honour to request you to kindly release one hundred pounds of quinine through Messrs. The Anglo-Thai Corporation, Karachi, for the bonafide use of the medical practitioners in Karachi. The medical practitioners have to undergo great difficulties in obtaining quinine for their patients as it is not available in the market here. The price of whatever stock that may be available is so fluctuating that in some cases it becomes prohibitive for the poor patients who are subjects of that dire disease, malaria.

Under the circumstances, I request that you will be kind enough to issue immediate orders to Messrs The Anglo-Thai Corporation to release at least one hundred pounds of quinine for the use of the members of the Karachi Medical Association (Sind Provincial Branch of the I.M.A.) and other members of the medical profession.

* * * * *

Copy of the letter No. 45/25/42/14-SI(II) dated the 24th July, 1942, from the Deputy Director General, Indian Medical Service (Stores) to the President, Karachi Medical Association (Sind Provincial Branch of I.M.A.):

With reference to your telegram dated the 20th July 1942, requesting this office to release 100 pounds of quinine sulphate from the quantity recently imported by the Anglo-Thai Corporation, Ltd., Karachi, for the use of the medical practitioners, it is understood that there are other dealers in Karachi who also hold stocks of this drug.

* * * * *

Copy of letter No. MC/13-2/36 dated 30th July, 1942, from Dr. R. A. Amesur, President, Karachi Medical Association (Sind Provincial Branch of the I.M.A.), Karachi, to the Deputy Director General, Indian Medical Service (Stores), New Delhi:

With reference to your Express letter No. 45|25|42|14-SI(II) dated the 24th July 1942, I have the honour to state that there are only two firms—Messrs The Anglo-Thai Corporation and Messrs The Volkart Bros.—which import quinine. The stock held by Messrs the Anglo-Thai has been frozen under the orders of the Government, while Messrs The Volkart Bros., who are the direct importers of "Roche" quinine, have no stock. The other local chemists when approached by the medical persons say that they have no quinine in their stock. From enquiries it is found that there are a few other sundry merchants, who have a small quantity of quinine for which they are quoting Rs. 156/- and round about that figure for a pound of quinine and even this they quote to non-medical persons for the reason best known to them. When medical persons approach them they say flatly that they have no quinine.

Owing to heavy rains in Karachi and very heavy floods in Sind, malaria has made its appearance in right earnest and it is becoming a serious problem to treat cases without quinine being made available to the medical profession. People who cannot afford to pay for their treatment will have to be sent to the Government and other Government-aided charitable institutions for the treatment. This will not only entail heavy expenditure to the Government but will also require more medical persons to treat and cope up with the rush of patients. Already Govern-

ment is handicapped about the medical recruits and this further requirement will create further difficulties.

As people from the flooded areas are coming to Karachi and there is a great liklihood of a great influx of such malaria infected people in Karachi it is very likely for the malaria infection to spread to the military areas to the detriment of the health and efficiency of the Army.

Under the circumstances, it is earnestly requested that the Government will be pleased to release the stock of quinine to the medical profession either through my Association or through Messrs The Anglo-Thai Corporation or any other Government agency, at cost.

WAR AND SUPPLY OF CERTAIN MEDICINES

The entry of Japan into the war has disturbed the supply of certain medicines, like menthol, camphor, agar-agar and quinine, to the allied countries. It has already been pointed out how the loss of Java, which produced 90 per cent of the world's quinine-containing bark, has caused a serious dislocation in the supply of quinine. Although the bark with the richest alkaloidal content (cinchona ledgeriana) does not take easily to Indian soils, still the areas already found to be suitable for the cultivation of other species of cinchona, if properly planted, can solve the difficulties to a great extent within the next few years. Many eyes are now turned towards India as one of the potential richest sources of medicinal drugs in the world. Podophyllum, belladonna, stramonium, liquorice, squill, colchicum, Karaya gum and valerian are easily obtainable and the lower slopes of the Himalayas offer fertile ground for further crops. A small quantity of ipecacuanha is being produced in Bengal and Madras, the largest bulk coming from Brazil. The war has shown how necessary it is to study the climatic and soil requirements of medicinal drugs in India, as also their active principles and physiological properties—*Science and Culture.*

INDIA-MADE SURGICAL INSTRUMENTS FOR RUSSIA

Nearly 80,000 surgical instruments manufactured in India have been supplied to the Indian Red Cross Commissioner for despatch to Russia. This was revealed at the 7th meeting of the Medical Stores Committee which met in New Delhi on July 10, with Lieut.-General Sir Gordon Jolly, Director General, I.M.S., in the chair. A representative of the drug manufacturers in India has now been included in the Committee.

Reviewing the progress of medical stores manufacture in India, the Committee noted that during the last six months 133 items have been transferred to list of medical stores article now made in India. Marked progress has been made in the production in India of acriflavine, atropine sulphate, catgut ligatures, tannic acid, creosote and ephedrine.

There has been important developments in the manufacture in India of surgical instruments and appliances, both as to variety and quantity, and the gross output of the industry has increased tenfold within the past twelve months. For example, one million "suture" needles were manufactured during six months ending with May, 1942, and the production of six million over the next 12 months has been planned.

BLOOD TRANSFUSION SERVICES IN GERMANY

A Ministry of the Interior circular dated the 5th March, 1940, lays down the terms of service for blood donors for the whole of Reich. It is estimated that one donor is required per 1,000 inhabitants, or 60,000 donors for the whole country. Since the number of hospitals is 3,000, each hospital has been instructed to have at its disposal about 20 donors. It has been advised that these donors should be in approximately the same proportions as those of the general population, *viz.,* O group 40%; A group 40%; B group 15%; AB group 5%.

Persons—men and women, who are between 21-50 years are asked to donate blood, provided they are found to be physically fit and are accepted only after a double examination of blood at recognised State Institutes. The donors, when accepted, carry a card, in different colours for each group, bearing their photographs and particulars of blood and hæmoglobin examinations. The donors receive 10 Rm. for the first 100 c.c., besides his cost of transport and compensation for loss of wages. These expenses are borne by the patient receiving the blood if he can afford to pay, or if he is insured, by his insurance society; if he is without means, they are met by the Government and local bodies.

As regards supplementary diet, blood donors, during the fortnight following the giving of blood, have a right to a supplementary ration of 250 gm. meat and 100 gm. of farinaceous foods per week, on condition that they have given at least 200 c.c. of blood—*Science and Culture.*

TREATMENT OF ELECTRIC SHOCK AND ELECTROCUTION

.....The Scientists have not yet discovered that electricity and magnetism are the male and female elements in the Universe.

Knowing as I do that everything in the Universe is composed of matter varying in vibration only, and that the spirit body is composed of ether, electric, and magnetic in its composition, one evening I inquired of one in the plane beyond the physical, one versed in the action of electricity, how it was that electricity could by its action destroy life, and I recall very distinctly his answer (Spirit of Michael Faraday).

"You are aware", he said, "of the voltage used in the various prisons when they put a criminal to death. You are also aware that frequently a current with many times the voltage used in electrocution passes through a body without serious injury. It may startle you to know that any person who has been electrocuted, or who has suffered a lightning shock, or who by accident has received a charge of electricity that has apparently produced death, could be restored to life by proper treatment. The charge of electricity, as applied in our prisons, paralyses the heart action, all the bodily functions, and the person is apparently dead. But you have—probably observed that whenever and wherever a person is put to death under sentence of the law, a post-mortem follows. Death was and is produced by the post-mortem and not by the electric shock. In the beginning surgeons were anxious to note the effect of the force, and undoubtedly made very careful post-

Continued at foot of page 36

BOOK REVIEWS

ARCHITECTURAL PRINCIPLES IN ARTHRODESIS—By H. A. Britan. 1942. Pages 132. Royal 8 vo. With 144 illustrations, eight in full color. Published by E. & S. Livingstone, 16 & 17, Teviot Place, Edinburgh. Price 21/- net.

Nowhere but in orthopœdics does one find so many variations in technique and operative procedures adopted by different authorities, in the light of their own experiences; nevertheless, the limitations of operative orthopœdic surgery with the ill-effects (both primary and secondary on a large number of "Failed" cases still remain painfully obvious.

The present author has taken recourse to sound principles of architecture and mechanics and the collaboration with his friend Mr. Humphrey Boardman for advice concerning architectural principles has been well sought indeed.

It is easy to recast or re-state established matters in a book form, but it demands ingenuity and unbiassed clear-thinking and experience to set out variations, by taking note of faulty principles of operations that lead to failure. Time and experience alone will show the efficacy of methods recommended but the results obtained by the author are truly encouraging.

A pleasantly written manual, admirably illustrated with photography and radiography, the paper, print and publishing being all excellent. Steps of operation are well illustrated and their purpose well explained with a fair amount of elaboration in details. Chapters on Arthrodesis of the hip, shoulder and knee are particularly well written. The book is warmly recommended to all those who are interested in the theory and practice of this special branch of surgery.

Amarnath Mukerji

Continued from page 35

mortems. You would be astounded to know, as we know, that post-mortems have lost interest and that frequently they now consist of jabbing a knife into the apparently dead body and passing it on for burial.

"When a person receives an excessive charge of electricity, either by accident or design, and the bodily functions are thereby temporarily paralysed, if the body were immediately stripped, laid upon the fresh earth and sprayed with water, the electricity would be drawn therefrom, and would pass into the earth. If then artificial movement of the arms and stimulants were resorted to, the heart action would be resumed, and one apparently dead would get up and walk away. Persons die from electric shock because they are not properly treated. When the bodily functions are paralysed and the electricity is not immediately drawn from the body and the action of the heart is not started by artificial means, death will, of course, ensue in a short time. If the treatment described is administered in time, there is no occasion for dissolution for electric shock. Electricity is life, and life will not destroy life. In this day, where electricity is in such common use, countless lives could be saved if the facts that I am now giving you were known and the treatment applied"—Extract from *The Dead Have Never Died* by Edward C. Randall, pp. 128-29, published by George Aleen & Unwin. Ltd., London.

ILLUSTRATIONS OF BANDAGING AND FIRST AID—By Lois Oakes, 1942. Second Edition. Pages vii+256. Demy 8 vo. Illustrated with over 300 photographs. Published by E. & S. Livingstone, 16 & 17, Teviot Place, Edinburgh. Price de luxe Edition 6/- net, paper Edition 4s. 6d. net, postage 6d.

The second edition of this valuable book is certain to be widely read and greatly appreciated, being easily the most valuable book on the subject presented in an interesting form in recent years. The different steps have been set out with almost mathematical simplicity. Illustrations neat and profuse, break-up pages of well printed text which is clearly brought out and thoroughly arranged. The excellence of turn out is typical of the publishers and certainly is above comment. We, however, recommend that in Section IV dealing with First-Aid in Fractures, the introductory portion be revised as the definition of fracture, its causation, points in recognition, etc., are rather unorthodox.

Amarnath Mukerji

WAR WOUNDS AND INJURIES—Edited by E. Fletcher and R. W. Raven with a foreward by Lord Horder, 1940. Pages viii+262. Demy 8 vo. Illustrated. Published by Edward Arnold & Co, London. Price 14/- net.

The book presents subjects of extreme topical interest of to-day. A distinguished team of contributors, many of whom have taken advantage of experience gained in the last Great War, provides the Medical Practitioner with authoritative yet unbiassed information on the most dependable and modern methods of management of War Wounds and Injuries. We have no hesitation in saying that the articles embodied will be of direct practical help to those for whom the book is intended.

Chapters dealing with injuries of many important abdominal organs as well as those on hæmorrhage, sepsis and burns are, however, greatly missed in the present edition, but we hope that they will be added on in the next.

Chapters dealing with wounds of the eye-ball, injuries of jaws and face and chest wounds are very usefully illustrated. Several of the chapters, especially those written by one of the editors, are appended with valuable references. The whole book is indeed well presented and the paper and print are both of the best.

The amount of useful information presented in so small a space is really amazing and does immense credit to the contributors and editors.

Amarnath Mukerji

DISEASES OF THE NOSE, THROAT AND EAR—By I. Simson Hall, 1941. Second Edition. Pages 446. Crown quarto. Published by E. & S. Livingtome, 16 & 17, Teviot Place, Edinburgh. Price 15/- net.

The author has succeeded in presenting in a compact form and in a practical manner the commoner diseases of the ear, nose and throat, met with in general practice and the book will be quite useful to the senior students and busy practitioners for whom it is intended; the chapter on endoscopy particularly will be read by them with profit. The publishers are to be congratulated for the excellent get up of the treatise.

Sachin Sen Gupta

CORRESPONDENCE

The Editor is not responsible for any views expressed by correspondents—EDITOR.

The Editor,
Journal of the I. M. A., Calcutta.

INDIAN MEDICAL COUNCIL AND ITS UTILITY

SIR,

Of the numerous indignities that a subservient nation has to suffer in the various spheres of human activity, the one that India was required to brook in 1933 was the foisting on her a legislation which brought into being a hitherto unwanted and now provenly futile body known as the Indian Medical Council. While in the political arena the behests of the Whitehall must be complicated with, in the field of medical legislation no less glorious a part was played by the General Medical Council of Great Britain and Ireland. It started active agitation in 1930 by refusing recognition to medical graduates from India, on grounds of inadequate clinical training and want of an acceptable standard of medical education. This resulted in an enquiry by Col. Needham, a retired officer of the Indian Medical Service who, as could only be expected, recommended as an essential prerequisite for recognition, the establishment of a central authority which could regulate medical education under the egies of the General Medical Council. In the interests of the young and ambitious aspirants for higher medical education, the only alternative for the Indian legislator was to place on the Statute Book an enactment now known as the Indian Medical Council Act of 1933. Under this Act a Council was established with the dual function of establishing a uniform minimum standard of higher medical education for the various British Indian Provinces and of negotiating reciprocal relations with foreign countries. This august body has not up-to-date been given the powers of maintaining an All-India Register, the one important function which similar institutions all over the world are required to perform, nor has it succeeded in eliciting General Medical Council's recognition for the qualifications granted by all the British Indian Universities, nor does its authority extend over the one third of this country, namely, Indian States. To add to it, the bulk of the medical profession —the Licentiates—are outside the jurisdiction of the Council, though thanks to the efforts of Mr. Kazmi, a bill for their inclusion is on the anvil of the legislature.

While central registration is yet a far cry, reciprocity with foreign countries in the midst of a world war is also a pious wish. Raising the standard of medical education to a higher uniform level by converting the existing schools into colleges or giving effect to the Council's recommendations having financial implications, is no less up-hill a task in the present circumstances, when all the revenues both central and provincial must, of necessity, be tapped for the war effort. These beneficent activities have naturally to take their turn after world conditions have returned to normality.

So much about the functions. One would naturally like to know about its finances. With no income of its own, the Council is solely dependent on annual grants by the Goverment of India. The administration of these funds is the concern of the Council subject to the general financial control of the donor. Huge sums doled out from the centre are sometimes spent, at others misspent. A detailed check on the Council's activities, not being either desirable or possible, it sometimes indulges in luxuries which one can illafford in these times. To mention only one of the several instances, the appointment of a retired officer as Secretary of the Council on a lavish four figure salary in 1937 and the recent decision to extend his tenure for another two years on the maximum of the scale, despite the physical disability which the advancing age naturally imposes on every living being, is a phenomina not easy for the sensible layman to comprehend. A body set up to regulate and improve medical standards should behave in such an unmedical fashion as to retain in its employ an over superannuated Secretary, is something not easy to explain. It must have been for none other than medical reasons that the maximum age of 55 years should have been fixed for retirement from Government service. Even persons with specialised knowledge and valuable experience such as the Judges of the High Court are not allowed to stay on beyond 60 for the simple reason that age does tell on the mind and the body after a certain stage. It is curious, therefore, that a body consisting entirely of experts in the various branches of medicine, should have thought it fit to give such a long rope of service to one who has even surpassed the maximum age limit fixed for the judges and should both in fairness to himself and the Council have long ago been ordered to complete rest under sound medical advice, yielding place to a much cheaper, but decidedly a more energetic and ambitious young man who would naturally be more qualified and better equipped both physically and mentally for secretariat work. Should the suspension of such a dormant body be considered outside the domain of practical politics, the Government and the Council should as well consider the possibility of having a part time Secretary till at least the duration of war. It is no less the concern of the Government as that of the taxpayer himself to see that public money is not thrown in such a wasteful channel when it is so badly required for strengthening the National War Front in so many directions. I am etc.

Dasan Street,
Delhi, 4-7-42.

S. C. ANAND,
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L.M.P. COURSE IN THE STATE MEDICAL FACULTY OF BOMBAY

Sir,

The decision of the Bombay Government to institute a State Medical Faculty to hold examination for a new diploma to be started by the Faculty and styled as L.M.P. (Bom.) was announced in the *Times of India* of the 3rd July 1942. The reason given for this decision of the Government was that the College of Physicians & Surgeons of Bombay would not go back on their decision to introduce the revised course for Licentiates.

This decision of the Government is deplorable as it is a definite retrograde step in the progress of medical education in the province. At a time when the Indian Medical profession and its various organisations including that of Licentiates, the Medical Council of India and the Central Government of India are trying

their best to raise the medical education to a uniform standard laid down by the Medical Council of India and the provinces are asked either to bring the standard of education in their medical schools to this uniform standard or to turn their schools into colleges as soon as possible, it is most unfortunate, nay, highly objectionable that the Bombay Government should take a definite step to lower the standard of education in the medical schools in the province, brought about by the revised regulations of the College of Physicians & Surgeons as a step towards bringing up the course of study in the medical schools, which aim the Government themselves have already proclaimed. The course now adopted by the Government amounts to going back not only on their own previous decisions but also on the recommendations of the Delhi Conference on the medical education, convened by the Government of India in November 1938, decisions to which their own Surgeon General at the time was a party. That Conference had recommended that one uniform standard of training and qualification for practitioners in modern scientific medicine should be established throughout India at an early date and that such standard shall be such as shall satisfy the requirements laid down by the Medical Council of India. Further, until the requirements of the Medical Council of India could be fully met, the Conference recommended that the improvements be effected in the existing medical schools regarding general preliminary education qualifying admission to medical schools, preclinical scientific subjects and clinical subjects.

One, therefore, fails to understand the object of the Government in their decision to institute an inferior medical diploma of L.M.P. One can only say that the Bombay Government intends to perpetuate the inferior standard of medical education in the province. This is quite clear, looking to the longdrawn struggle extending for nearly 15 years between the College of Physicians & Surgeons, Bombay, and the Bombay Government in the matter of raising the standard of education in the medical schools in Bombay Province.

In the year 1927, the College of Physicians & Surgeons of Bombay took up the question of raising the standard and approached the Government. Since then at the requests of the latter, the final action was deferred thrice by the College. In December 1934, the Government promised to make every effort to provide the necessary additional laboratory accommodation, equipment and staff by June 1939. Relying on this promise the College, in January 1937, raised the standard of preliminary qualification from Matriculation to Inter Science with Biology as the optional subject. The Government did not keep its promise. In 1938 it asked the College to postpone introduction of the revised course till 1940. In March 1940 it asked for another postponement till June 1941 as they were considering the question of converting the medical schools into colleges. In June 1940, a Conference of the representatives of the College and the Surgeon General was held and it was agreed that no admissions should be made after July 1940 for the L.C.P.S. Course in medical schools at Poona, Ahmedabad, Hyderabad (Sind), Miraj and National Medical College, Bombay, and after December 1940 into K.E.H. Medical School, Indore. As nothing was heard from the Government till 1941 January, inquiries were made from it by the College as to their decision on the subject. In February 1941, the Government informed the College and it was opposed to immediate stoppage of admissions to the schools and even to introduction of the revised regulations by the College. There seemed to be no action taken by the Government to convert the schools into colleges. I leave it to the medical profession and the public to judge the Government actions and their intentions in the matter.

In the meantime, the Madras and U P. Governments converted their medical schools into colleges and abolished L.M.P. diploma. The Punjab Government raised the standard of education in their medical schools.

Under the circumstances given above and with the stubborn attitude of the Bombay Government not to raise the standard of education in the medical schools in the province, the College had to take the final decision of introducing its revised course of 4½ years from June 1941.

This was the only decision that the College could have taken in the interest of the students, the profession and the public, to comply to a great extent with the recommendation of the Delhi Conference and to bring the standard of L.C.P.S. (Bom.) very near to one laid down by the Medical Council of India. One could not understand in whose interest the Government took this retrograde step. It is neither in the interest of the students because the low standard will debar them from any future prospect of going up for higher studies and the public would have inadequately qualified medical practitioners. The previous Surgeons General had all opined in favour of improving the L.C.P.S. course. Then why this sudden somersault on the part of Government on this occasion?

I must say that 4½ years could have been better distributed between the various examinations. If the period of preclinical studies was spread over two years, the 2nd step toward reaching the uniform standard might have been reached, the first being preliminary education upto Inter Science already introduced. But this was a matter of details and adjustment.

I may state that to help the Licentiates, the College has revised the M.C.P.S. course which can be now taken by a 4½ years course Licentiate by appearing straight at the Final M.C P.S. Examination after one year's Post Graduate training. M.C.P.S. (Bom.) is recognised by the Medical Council of India and also by the Government of India in its recent announcement of throwing open to them the Emergency Medical Commission (I.M.S.). The University has also allowed the M C.P.S. to appear at M.B.B.S. examinations without keeping term at the College.

One wonders how the President of the Bombay Branch of the Licentiates' Association can hail the retrograde step of the Government which will perpetuate the inferior medical qualification in the province. The All-India Licentiates' Association and its Branches throughout the country have cried themselves hoarse day after day for years to remove the stigma of inferior qualification while the President, Bombay Branch, rejoices at the perpetuation of it.

The Government action of instituting a new inferior diploma of L.M.P. (Bom.) deserves condemnation by the medical profession and I am sure the medical organisation in the province and the public will strongly protest against putting the Government decision into action. I am etc.

CHAMANLAL MEHTA

Bombay, 31-8-42.